United States Court of Appeals

for the Second Circuit

APPENDIX

76-6075

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United States Court of Appeals
FOR THE SECOND CIRCUIT

Docket No. 76-6075

NATURAL RESOURCES DEFENSE COUNCIL, INC.; NEW YORK ASSOCIATION FOR BRAIN INJURED CHILDREN; CENTER FOR SCIENCE IN THE PUBLIC INTEREST; ENVIRONMENTAL ACTION COALITION; FRIENDS OF THE EARTH; HIGHWAY ACTION COALITION; NATIONAL WELFARE RIGHTS ORGANIZATION,

Plaintiffs-Appellees,

—v.—

RUSSELL TRAIN, as Administrator of the U.S. Environmental Protection Agency; and the U.S. ENVIRONMENTAL PROTECTION AGENCY,

Defendants-Appellants.

ON APPEAL FROM THE UNITED STATES DISTRICT COURT FOR THE SOUTHERN DISTRICT OF NEW YORK

JOINT APPENDIX OF PLAINTIFFS-APPELLEES AND DEFENDANTS-APPELLANTS

ROBERT B. FISKE, JR.,
United States Attorney for the Southern District of New York,
Attorney for Defendants-Appellants,
One St. Andrew's Plaza,
New York, New York 10007.

DAVID SCHOENBROD
Attorney for Plaintiff-Appellees
15 West 44th Street
New York, New York 10036

UNITED STATES COURT OF APPEALS
FILED
JUL 16 1976
A. DANIEL FUSARO, CLERK
SECOND CIRCUIT

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JUDGE

CIVIL DOCKET

UNITED STATES DISTRICT COURT

A 1

Jury demand date:

74 Civ. 4617

Form No. 106 Rev.

TITLE OF CASE	ATTORNEYS
NATURAL RESOURCES DEFENSE COUNCIL, INC. NEW YORK ASSOCIATION FOR BRAIN INJURED CENTER FOR SCIENCE IN THE PUBLIC INTEREST ENVIRONMENTAL ACTION COALITION FRIENDS OF THE EARTH HIGHWAY ACTION COALITION NATIONAL WELFARE RIGHTS ORGANIZATION v RUSSELL TRAIN, as Administrator Environmental Protection Agency U.S.ENVIRONMENTAL PROTECTION AGENCY	For plaintiff: NATURAL RESOURCES DEFENSE COUNCIL INC. 15 W. 44th St.,N.Y.10036 - 869-0150 For defendant: U.S. Atty.

STATISTICAL RECORD	COSTS	DATE	NAME OR RECEIPT NO.	REC.	DISB.
. 5 mailed x	Clerk	10/3/74	Cert Mail Rec'd	15 —	
		10/3/74	In Stamps		15 —
S. 6 mailed	Marshal				
sis of Action: Declaratory Injunctive relief - Citizens Suit Section Clean Air act	Docket fee				
	Witness fees				
tion arose at:	Depositions				

ET AL.

NATURAL RESOURCES DEF. COUNCIL V. RUSSELL TRAIN ADM., ETC.

74 CIV. 20

A 2

DATE	PROCEEDINGS	Date Order Judgment N
.21-74	Filed complaint and issued summons.	
31-74	Filed summons with marshal's return. Served on; Paul Curray,US Atty., by P. Troia on 10/22/74 Atty. Gen.,Wash.,DC by certified mail #162225 on 10/22/74	
.21-74	Filed affdt. and notic e of motion by Exxon Corp. for an order granting leave to said party to intervene as a party deft, etc. as indicated,ret. on: Dec. 5,1974 at 10am in Rm.1305.	
.21-74	Filed memorandum of law in supprt of motion to intervene by Exxon Corp.	
.26-74	Filed pltfs. affdt. and notice of motion for summary judgment in favor of pltfs. pursuant to rule 56,for declaratory judgment and mandamus etc.ret. on: Dec. 16,1974 at 4pm in Rm.1305.	
.26-74	Filed pltfs. memorandum of law in support of ptlfs. above motion.	
.26-74	Filed pltfs. statement of material facts as to which there areno germane issues of fact to be tried.	
v.29-74	Filed stip. and order that Exxon Corp. shall forthwith withdraw its motion to intervene and covenants not to file any similar motion in this action,subject to pltfs. compliance with the providions of paragraph 2;pltf. shall file an amended complaint striking the following allegations of the complaint as indicated,etc. as indicated. So ordered,Stewart,J. (J.)	
.5-74	Filed order--At the request of pltfs, the following allegations of the amended complaint dated Nov. 27,1974 are hereby striken to correct clerical errors,etc. as indicated. So ordered,Stewart,J. m/n	
c.6-74	Filed Brief of Amicus Curiae, the City of New York.	
c.3-74	Filed pltfs. amended complaint.	
.17-74	Filed defts. affdt. and notice of motion for an order to dismiss complaint or in the alternative,for summary judgment,etc. ret. on: Dec.18,1974.	
ec.17-74	Filed defts. memorandum of law in support of motion to dismiss,etc.	
c.24-74	Filed defts. reply memorandum of law.	
	========= ========= ========= error =========	
03-02-76	Filed OPINION #43978...Defendants moved to dismiss for lack of jurisdiction; failure to state a claims and summary judgment. Plaintiffs have also moved for summary judgment. ..... The Court has jurisdiction. For reasons stated herein, it is ordered that the Administrator place lead on the list of pollutants, in accordanc with the mandate of §108, within 30 days from the date of this decision. So ordered. -- Stewart, J. m/n	
4-29-76	Filed defts notice of appeal to the U.S.C.A for the Second Circuit from the order entered on 3-1-76. Mailed copy to David Schoenbrod.	
6-02-76	Filed defts brief.	
06-02-76	Filed pltf reply memorandum of law	
6-02-76	Filed letter by the U.S. Atty;s to Judge Stewart encloses copies of performance Standards of Phosphate Fertilizer Industry.	

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U.S. DISTRICT COURT
FILED
MAR 2 1976
S.D. OF N.Y.

UNITED STATES DISTRICT COURT
SOUTHERN DISTRICT OF NEW YORK

- x

1-3313

NATURAL RESOURCES DEFENSE COUNCIL, INC., et al.,

Plaintiffs,

-against-

74 Civ. 4617

RUSSELL TRAIN, Administrator Environmental Protection Agency, et al.,

Defendants.

#43978

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M E M O R A N D U M

STEWART, DISTRICT JUDGE:

Natural Resources Defense Council, Inc. ("NRDC") and other named plaintiffs bring this action against the Environmental Protection Agency ("EPA") and its administrator Russell Train for failure to list lead as a pollutant under §108 of the Clean Air Act of 1970. Defendants have moved to dismiss the complaint for lack of jurisdiction and failure to state a claim or for an order granting summary judgment. Plaintiffs have also moved for summary judgment.

Plaintiffs have alleged four separate grounds upon which the court might find jurisdiction: 1) §304 of the Clean Air Act, as amended, 42 U.S.C. §1857h-2(a); 2) the Administrative Procedures Act, 5 U.S.C. §701-706; 3) the Declaratory Judgment Act, 28 U.S.C. §§2201-2; and 4) the mandamus provisions of 28 U.S.C. §1361.

Section 304 of the Clean Air Act provides in pertinent part:

> Any person may commence a civil action on his own behalf (2) against the Administrator where there is alleged a failure of the Administrator to perform any act or duty under this chapter which is not discretionary with the Administrator.

Defendants argue that the listing of pollutants under §108 is a discretionary function and therefore no jurisdiction is vested in this court by virtue of §304. While §304 does not provide jurisdiction over distinctly discretionary functions of the Administrator, see e.g., United States Steel Corp. v. Fri, 364 F. Supp. 1013 (N.D. Ind. 1973), it does permit jurisdiction to decide whether a function is mandatory or discretionary. Cf. Bell v. Hood, 327 U.S. 678 (1945).[1/] We therefore do not need to consider plaintiffs' other asserted jurisdictional grounds.

We turn now to the merits of plaintiffs' claim. Section 108 provides that the Administrator shall publish, and from time to time revise, a list including each air pollutant

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1/ Plaintiffs essentially seek a writ of mandamus to compel performance of the alleged duty to list lead as a pollutant under §108. Mandamus is an appropriate remedy under §304 of the Clean Air Act to compel performance of a non-discretionary duty. See NRDC v. EPA, D.C. Cir. No. 72-2233 (1973); Citizens Association of Georgetown v. Washington, 370 F. Supp. 1101 (D.D.C. 1974).

> (A) which in his judgment has an adverse effect on public health or welfare;
>
> (B) the presence of which in the ambient air results from numerous or diverse mobile or stationary sources; and
>
> (C) for which...he plans to issue air quality criteria under this section.

Plaintiffs contend that the statutory language, legislative history and purpose, as well as current administrative interpretion of the 1970 Clean Air Act, all militate in favor of finding that the Administrator's function to list pollutants under §108 is mandatory, once it is determined by the Administrator that a pollutant "has an adverse effect on public health or welfare" and comes from the requisite numerous or diverse sources. Defendants concede in this action that lead comes from the requisite sources and that the Administrator has found lead to have the required "adverse effect." Defendants argue, however, that the language of §108(a)(1)(C) "for which...[the Administrator] plans to issue air quality criteria" is a separate and third criterion to be met before §108 requires placing a pollutant on the list. This construction of §108(a) leaves the initial decision to list a pollutant within the sole discretion of the Administrator. Defendants contend such discretion is required because the Administrator must choose between alternative remedies provided in various sections of the Act and that any decision to utilize the

remedies provided by §§108-110 "involves complex considerations." (Defendants' brief at 22).

Through analysis of the parties' arguments and of the various remedies and provisions of the Act, we have determined that the statutory scheme contemplates a mandatory duty on the part of the Administrator which is enforceable in the instant action.

Congress, in passing the Clean Air Act of 1970, was concerned with the delays and inefficiencies incurred in implementing the 1963 and 1967 air pollution acts. The House Report stated that "progress [had] been regrettably slow," citing "cumbersome and time-consuming procedures called for under the 1967 Act," "organizational problems on the federal level where air pollution control has not been accorded a sufficiently high priority" and "failure on the part of the National Air Pollution Control Administration to demonstrate sufficient aggressiveness in implementing present law." HR. REP. 91-1146, 91st Cong., 2d Sess. 5 (1970). Thus, in the language of the 1970 Act, Congress attempts to achieve cleaner air by specifying procedures and timetables to be followed, all of which are reflective of Congress' determination "to speed up, expand, and intensify the war against air pollution in the United States." HR. REP. 91-1146, 91st Cong., 2d

Sess. 1 (1970).[2/]

Defendants' main argument in support of its construction of the section as discretionary is that there are other sections within the Act which provide alternative remedies for lead pollution. The existence of alternatives, defendants contend, requires that the Administrator should have the discretion to choose among the remedies provided by the Act.

In addition to the explicitly stated purpose of the Act, as outlined above, we do not think that the statutory language supports defendants' construction of the Act. There is no language anywhere in the statute which indicates that the Administrator has discretion to choose among the remedies which the Act provides. Rather, the language of §108 indicates that upon certain enumerated conditions, one factual and one judgmental, the Administrator "shall" list a pollutant which triggers the remedial provisions of §§108-110. The statute does not provide, as defendants would have it, that the Administrator has authority to determine whether the statutory remedies

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2/ In this statutory scheme, the listing of a pollutant under §108(a)(1) triggers the setting of air quality criteria within 12 months after that listing (§108(a)(2)) and compels issuance of information about air pollution control techniques (§108(b)(1)) and establishment of national primary and secondary ambient air quality standards for each pollutant. (§109(a)(2)). Finally, within 9 months of the Administrator's setting the national ambient air quality standard, each state must submit a plan through which the state can meet that standard. (§110(a)).

which follow a §108 listing are appropriate for a given pollutant.

We think the reasonable reading of the disputed language in §108 is that the Administrator must include on the initial list to be issued 30 days after December 31, 1970, all those pollutants "for which air quality criteria had not been issued before [that date]" but which pollutants he has already found in his judgment to have an adverse effect on public health or welfare and to have come from the requisite sources.[3/] The Senate Committee Report supports our reading of the language. The Report states that §108 requires the initial list to "include all those pollution agents which have, or can be expected to have, an adverse effect on health and welfare and which are emitted from widely distributed mobile and stationary sources, and all those for which air quality criteria are planned." S. REP. No. 91-1196, 91st Cong., 2d Sess. 54 (1970). It is to the initial list alone that the phrase "but for which he plans to issue air quality criteria" is directed; the phrase

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3/ Our construction of the statutory language renders irrelevant arguments by both sides concerning the five pollutants which the senate subcommittee had examined. We do not think that Congress intended to bind the Administrator to list any particular pollutants but only to list those pollutants which met the two criteria established in the statute. Since the Administrator has either found that the other pollutants considered by Congress do not have an "adverse effect" or do not come from the requisite sources, those pollutants are not in the same category with lead which concededly meets the two requirements of the section.

cannot mean that the Administrator need not list pollutants which meet the two requisites clearly set forth in the section.[4/] Again, that construction would comport with neither the clear legislative intent to have strict mandatory health procedures in effect by mid-1976 nor the language of the Act itself. While the Administrator is provided with much discretion to make the threshold determination of whether a pollutant has "an adverse effect on health," after that decision is made, and after it is determined that a pollutant comes from the necessary sources, there is no discretion provided by the statute not to list the pollutant. We think that Congress intended to trigger the elaborate procedures of §§108-110 whenever the above two factors were found to exist.

Although we have found no authority which has directly confronted this question, §108 has been construed to require listing of all pollutants for which the two enumerated criteria have been met. That was the interpretation initially placed upon the section by the Administrator on January 30, 1971.

4/ Further, we note that defendants' construction of §108 would render the mandatory issuance of a list meaningless. Under defendants' construction of §108, it might well be that the Administrator would choose not to list any pollutant at all, but would nevertheless be required by the legislation to issue a "list." We cannot find support for such a construction.

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36 Fed. Reg. 1515 (1971). That is the same construction which has been placed upon the section by other courts in dicta. See Indiana & Michigan Electric Co. v. EPA, 509 F.2d 839, 841 (7th Cir. 1975); Kennecott Copper Corp. v. EPA, 462 F.2d 846, 847 (D.C. C. 1972).

With specific reference to the lead pollution at issue here and in support of their position that the Administrator should have discretion under the Act to choose among remedies provided by various sections, defendants point us to the Administrator's decision to regulate lead in gasoline under §211(c)(1)[42 U.S.C. §1857f-6(c)(1)].[5/] Defendants assert that the EPA considered regulating lead pollution either by setting national ambient air standards under §108 or by establishing standards for the lead content of motor vehicle gasoline under §211.[6/] However, as we read the two statutory sections, §108 and

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5/ Section 211(c)(1) provides, in pertinent part, that
The Administrator may, from time to time...control or prohibit the manufacture, introduction into commerce, offering for sale, or sale of any fuel or fuel additive for use in a motor vehicle or motor vehicle engine (A) if any emission products of such fuel or fuel additive will endanger the public health or welfare, or (B) if emission products of such fuel or fuel additive will impair to a significant degree the performance of any emission control device or system which is in general use, or which the Administrator finds has been developed to a point where in a reasonable time it would be in general use were such regulation to be promulgated.

6/ In fact, the Administrator did set regulations under §211 for lead in gasoline which were subsequently struck down by the Court of Appeals for the District of Columbia. A rehearing en banc was granted and the case is now sub judice before the full court. Ethyl Corp. v. EPA ___ F.2d ___(D.C.C.), reh. en banc ___F.2d___ (1975).

and §211, they are neither mutually exclusive nor alternative provisions. Defendants' argument is premised upon the misconception, as discussed above, that the statutory scheme provides alternative remedies for pollutants. We think the misconception becomes clear through analysis of the specific case of lead pollution regulation.

Defendants state the reasons for the Administrator's decision to regulate lead under §211.

> It was the Administrator's judgment that (a) uniform standards would make industry compliance simpler than a proliferation of differing state standards, (b) federal controls at the refinery level would be more efficient than state or local controls directed at thousands of distributors and retailers, and (c) the states were hard-pressed to implement the six existing ambient air quality standards and should not be assigned another major regulatory task when equally effective alternatives were available.

These reasons could have no bearing upon a policy decision, if one were permissible under the Act, of whether or not to establish national standards under §108. The benefits of uniform standards, of federal controls and of averting "another major regulatory task" for the states can all accrue from regulations under §211 when lead is listed under §108. If the Administrator sets standards under §211 which effectively decrease the level of lead in the ambient air by taking lead out of gasoline, then the decrease in pollution brought about by the §211 regulations will be taken into account by each state when it submits its

plan to meet the national standard set under §110.[7] In fact, §211(c)(4)(C) provides that the only circumstances under which the Administrator may approve the regulation of motor vehicle fuel or fuel additive in an implementation plan promulgated pursuant to §108 is where "he finds that the state control or prohibition is necessary to achieve the national primary or secondary ambient air quality standard which the plan implements."[8] Therefore, if the Administrator had made the necessary regulations under §211, he cannot permit the states to regulate lead emissions through the implementation plans provided for in §110. In the event that regulation under §211 causes a decrease in air lead sufficient to meet any national standard set under §108, then there will be uniform standards, federal regulation and no further regulatory task for the states. Despite regulation under §211, however, the Administrator must nevertheless list lead as a pollutant since it concededly meets the two criteria of §108 and must set in motion the collection of scientific data and the issuance of criteria and national standards. We believe it is necessary to "literally comply with

~~Further,~~

7/ Plaintiffs are correct in stating that no state controls are required where the ambient air standard is achieved through federal emission limitations under section 202[42 U.S.C. §1857f-1]. 40 CFR §51.14.

8/ We cannot agree with defendants' contention that §211(c)(4)(C) "supports EPA's construction" of §108. (Defendants' reply memorandum at 2). We do not think the cited section supports either parties' position with regard to the mandatory or discretionary language of §108.

all the formal steps provided by the statute" even if "the extent to which compliance with these requirements might produce beneficial results is not known." NRDC v. EPA, 475 F.2d 968, 972-3 (D.C. C. 1973) (MacKinnon, J., concurring). One reason to enforce compliance is seen in the disagreement on the comparative rigors of the "adverse effect" standard in §108 and the "will endanger" standard in §211. The only agreement is that these two phrases represent different standards. Ethyl Corp. v. EPA, ___ F.2d ___ (D.C.C.) reh. en banc ___F.2d ___ (1975). It is conceivable, therefore, that regulation of lead under whichever one of these sections is characterized as less stringent would not be sufficient to meet the more rigorous standard of the other section. The acknowledged difference in the two standards is further support for our finding that these two provisions were not meant to be alternative remedies for pollutants.

Finally, we turn to an additional argument of defendants that the Administrator needs discretion not to list lead under §108 because the data which would be necessary to support an ambient air standard for lead is arguably lacking. Defendants concede, however, that such a potential lack of data did not enter into the Administrator's decision not to list lead at the time it was made. We do not think that the potential lack of data would have been an appropriate consideration prior

to listing a pollutant under §108 in any event. Under the statutory scheme, the listing of a pollutant is no more than a threshold to the remedial provisions. Before a listing, the statute provides for the Administrator to exercise his judgment concerning whether or not a pollutant, here lead, has an adverse effect upon the public health. Once he has made that judgment, however, the Administrator does not have discretion not to list lead as a pollutant because necessary data---data other than that necessary to make the initial decision as to "adverse effect"---is unavailable. The statute appears to assume that, for each pollutant which must be listed, criteria and a national standard can be established. A twelve month period is provided for that purpose. However, Congress cannot require the impossible. It may be that a pollutant exists which meets the listing requirements of §108 but for which no criteria or national standard is possible. That issue is not before this court.[2/] The only question here is the threshold one of whether lead must be listed according to §108 and we have determined that it must.

2/ Congress did contemplate criteria might be set with which it would be technically unfeasible to comply. Section 110(e) provides for extensions in the time for compliance in such instances. See NRDC v. EPA, 475 F.2d 968 (D.C. Cir. 1973). Congress did not provide, however, that criteria should not be set at all.

We cannot find support for defendants' position that there is "much to be lost by limiting EPA's ability to select from its sources of authority in the Clean Air Act the control strategy best suited to the pollution problem at hand." (Defendants' memorandum in support of motion to dismiss at pp. 20-21). We are convinced that the Administrator has considerable, and sufficient, discretion. Not only does he exercise his judgment over the initial determination of whether a pollutant "has an adverse effect upon health," but also he must set the national standard "which in [his] judgment...is requisite to protect the public health." (§109(b)(1)). Finally, the Administrator must approve the implementation plans of the states. We think that these provisions give the Administrator all the discretion which Congress contemplated that he have.

In the instant case, the Administrator has conceded that, in his judgment, lead "has an adverse effect on health" and comes from "numerous or diverse mobile or stationary sources." The two statutory criteria having been met and this court having determined that a duty thereafter arises, it is

ORDERED that the Administrator place lead on the list of pollutants, in accordance with the mandate of §108, within 30 days from the date of this decision

SO ORDERED.

United States District Judge

Dated: New York, N. Y.
March / , 1976.

A 16

UNITED STATES DISTRICT COURT

FOR THE SOUTHERN DISTRICT OF NEW YORK

NATURAL RESOURCES DEFENSE COUNCIL, INC.
15 West 44th Street
New York, New York 10036

NEW YORK ASSOCIATION FOR BRAIN INJURED CHILDREN
95 Madison Avenue
New York, New York

CENTER FOR SCIENCE IN THE PUBLIC INTEREST
1779 Church Street, NW
Washington, D.C.

ENVIRONMENTAL ACTION COALITION
1346 Connecticut Avenue, NW
Washington, D.C.

FRIENDS OF THE EARTH
620 C Street, SE
Washington, D.C.

HIGHWAY ACTION COALITION
Room 731, DuPont Circle Building
Washington, D.C.

NATIONAL WELFARE RIGHTS ORGANIZATION
1420 N Street, NW
Washington, D.C.,

Plaintiffs,

v.

RUSSELL TRAIN, as Administrator
Environmental Protection Agency
4th & M Streets, SW
Washington, D.C.

U.S. ENVIRONMENTAL PROTECTION AGENCY
4th & M Streets, SW
Washington, D.C.,

Defendants.

CIVIL ACTION
NO. 74-4617

AMENDED COMPLAINT

I. INTRODUCTION.

This case is about airborne lead pollution, which Defendants EPA Administrator Russell Train and EPA have officially found to endanger the physical and mental health of people, particularly city children. But, the Defendants have refused to carry out their clear statutory duty to treat lead under the health

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United States Attorney
U. S. Courthouse
Foley Square
New York, N. Y. 10007

protection procedures of the Clean Air Act. Sections 108-110*. These procedures require the Administrator to promulgate a national primary ambient air standard for lead, requisite to protect health, and to attain this standard no later than May 29, 1976. The Administrator's refusal to perform these mandatory duties will also result in the preemption of New York City's lead in gasoline ordinance on January 1, 1975, thereby tripling the lead content of gasoline sold in the City. Section 211(c)(4)(C); Exxon Corp. v. City of New York, 73 Civ. 1047 (S.D.N.Y. Order of March 8, 1974).

Plaintiffs, organizations concerned with children's health and the environment, bring this action under the Citizens Suit Section of the Clean Air Act, to compel the treatment of lead under the Act's health protection procedures and, accordingly, to prevent the preemption of the City's lead-in-gasoline ordinance on January 1, 1975.

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* The Clean Air Act is codified as 42 U.S.C. §1857 et seq. The sections of the Act relied upon herein are:

Section 108	42 U.S.C. §1857(c)(3)
Section 109	42 U.S.C. §1857(c)(4)
Section 110	42 U.S.C. §1857(c)(5)
Section 211	42 U.S.C. §1857f-6c
Section 304	42 U.S.C. §1857h-2

Since the code citation becomes confusing, especially when subsections and subparagraphs are cited, this Complaint hereinafter will cite to the Act.

II. PARTIES.

A. Plaintiffs.

1 The Natural Resources Defense Council, Inc. [NRDC] is a non-profit, membership corporation organized under the laws of the State of New York. NRDC's principal office is at 15 West 44 Street, New York, New York 10036. It also has offices in Washington, D.C., and Palo Alto, California. NRDC has 12,000 members and contributors throughout the country, of whom 1,800 live in New York City. NRDC's purposes, which are supported by its members and contributors, are to enhance and protect environmental quality, to monitor federal, state, and city agencies responsible for implementing environmental legislation, and, when necessary, to litigate the adequacy of agency actions implementing such statutes.

2 The New York Association for Brain Injured Children [NYABIC] is a not-for-profit corporation formed under the laws of the State of New York. NYABIC and its local chapters have 3,200 member families, who are virtually all parents of brain injured children. The NYABIC chapters in New York City have a total membership of 1,100 families. These members of the New York City chapters live within the City of New York.

Brain injury is a neurological disorder, which impairs the ability of children with normal intelligence to learn. Its symptoms are hyperactivity, inability to concentrate, poor impulse control, and difficulty learning to read and write. Lead poisoning, which children can contract by eating lead paint and lead-laden dust, and by breathing ambient lead is a major cause of brain injury.

NYABIC's purpose as an organization is to promote the interests of brain injured and learning disabled children, both

by obtaining better services for those children who already suffer the consequences of brain damage and by preventing future brain damage to other children. In furthering its purposes, NYABIC has worked with government agencies to provide more educational and medical services to brain injured children. It currently operates three schools to provide special education to children with the learning disabilities associated with brain injury. It also provides the general public with information about brain injury, its symptoms and causes, including lead poisoning.

3 The Center for Science in the Public Interest is a not-for-profit corporation organized under the laws of the District of Columbia and has its main office at 1779 Church Street, NW, Washington, D.C. Its purpose is to provide scientific information on issues of public importance and to promote adequate government safeguards of the public health.

4 Environmental Action Coalition is a not-for-profit corporation organized under the laws of the District of Columbia and has its main office at 1346 Connecticut Avenue, NW, Washington, D.C. 20036. It has approximately 7,500 supporters. Its purposes are to protect the environment and safeguard human health.

5 Friends of the Earth is a not-for-profit corporation organized under the laws of the State of New York, which has its main office in San Francisco, California, and a branch office at 620 C Street, SE, Washington, D.C. It has 28,000 members throughout the country. Its purposes are to preserve the human environment and protect human health from pollution.

6 The Highway Action Coalition is composed of approximately 20 membership organizations. It has its main office at Room 731, DuPont Circle Building, Washington, D.C. In addition to the 20

organizations which form the coalition, Highway Action has 3,000 direct supporters. Its purpose is to promote development of sane transportation policies in the United States and to oppose the harmful environmental effects of extensive use of automobiles.

7 The National Welfare Rights Organization [NWRO] is a coalition of 40 state chapters located in 40 different states, including New York, and having a total membership of 16,000 individuals. The New York State chapter has members living in New York City. The national organization has its offices at 1420 N Street, NW, Washington, D.C. 20005. The purpose of NWRO and its chapters is to protect the rights and interests of poor people, particularly those who are dependent on public assistance. It is particularly interested in preventing lead poisoning from any source. Its members and their children, like all poor people, are victimized by lead poisoning more than the population as a whole, because they frequently live in urban areas where ambient lead, lead-laden dust and lead based paint abound.

B. Defendant.

8 Russell Train is the Administrator of the Environmental Protection Agency of the federal government and, as such, is responsible for the administration of that agency in accordance with the Clean Air Act, 42 U.S.C. §1857 et seq., and the Administrative Procedure Act, 5 U.S.C. §500 et seq.

9 The U.S. Environmental Protection Agency is the agency of the federal government responsible for administering the Clean Air Act, 42 U.S.C. §1857 et seq.

III. JURISDICTION, VENUE, NOTICE.

10 The jurisdiction of this Court is invoked under section 304 (42 U.S.C. §1857h-2)(citizen suits to compel the Administrator to perform non-discretionary duties); 28 U.S.C. §1361 (action to compel an officer of the United States to perform his duty); 5 U.S.C. §701-706 (review of federal administrative actions); and 28 U.S.C. §2201-2, F.R.C.P. §57 (declaratory judgment).

11 Venue of this action lies in the U.S. District Court, in the Southern District of New York by virtue of 28 U.S.C. §1391 (e)(4).

12 EPA was given notice of the failure to list lead pursuant to section 108(a)(1) and the demand to do so in the following communications:

a. Comments on EPA's Proposed Regulations on Lead Additives and Petition for the Issuance of Air Quality Criteria and National Ambient Air Quality Standards for Lead, David Schoenbrod, Louis Slesin, Natural Resources Defense Council, Inc. (March 18, 1973).

b. Supplemental Comments on EPA's Proposed Regulations on Lead Additives and Supplemental Petition for the Issuance of Air Quality Criteria and National Ambient Air Quality Standards for Lead, David Schoenbrod, NRDC, (May 15, 1973).

c. Letter from David Schoenbrod to Robert Sansom, Assistant Administrator, Air and Water Programs, EPA (March 14, 1973).

d. Letter from David Schoenbrod to Robert Sansom, Assistant Administrator, Air and Water Programs, EPA, (May 16, 1973).

e. Brief in Chief for Petitioners in NRDC et al. v. EPA, No. 74-1023 (D.C.Cir. April 10, 1974).

f. Reply Brief of Petitioners in NRDC et al. v. EPA, No. 74-1023 (D.C.Cir. July 29, 1974).

g. Letter from David Schoenbrod to Russell Train, Administrator, EPA (October 2, 1974).

Each of these communications was received by EPA and each contains all of the information required by 40 C.F.R. §54.3(a). The Admin-

istrator has explicitly refused to comply with these demands. 38 Fed. Reg. 33734, 40 (Dec. 6, 1973).

IV. STATEMENT OF THE CASE.

A. The Health Effects of Airborne Lead.

13 EPA acknowledges the toxicity of airborne lead and has found that:

a. Lead poisoning results in death or severe illness, including irreversible brain damage. EPA's Position on the Health Implications of Airborne Lead at III-1 to 5 (Nov. 28, 1973).

b. Lead exposure insufficient to cause overt symptoms alters vital bodily functions, such as the production of hemoglobin. Id. at III-9 to 10. On the basis of such effects, EPA has defined as unacceptable blood lead levels (id. at III-11), which are commonly exceeded. Id. at VII. While medical science has done little to investigate these alterations in bodily functions (id. at III-3), such subclinical exposure has been associated in children with subtle damage to the nervous system (including hyperactivity and loss of motor control), kidneys, and other systems. Id. at III-5 to 11.

c. Airborne lead has resulted in street dust in cities, such as Washington, D.C., and Boston, containing up to one percent lead (id. at II-6), equivalent to dangerous lead paint.

d. A quarter of urban children tested and millions of adul[illegible]e unacceptable blood lead levels due to lead additives. Id. a[illegible]-Tables. Humans absorb airborne lead from air breathed, water and food consumed, and dust and dirt pervading the metropolitan environment. Id. at V, VII, VII-3. This absorbtion, on top of naturally occurring lead in food, causes unacceptable

blood lead levels in the children, "a small, but significant" portion of all adults, and an important portion of the large groups of workers, such as garage mechanics and policemen, occupationally exposed to car fumes. Id. at VII and Tables.

e. Airborne lead is implicated in clinical lead poisoning. "For a child with pica for paint, the combination of the ingestion of a few chips of paint and an increased intake of lead from contaminated dusts would provide a total lead intake sufficient to cause systematic illness." Id. at VI-6. In addition, as many as half the cases of childhood lead poisoning cannot be traced to lead paint (id. at VI-12 to 13), so that lead dusts may be "an important contributing factor." Id. at VI-13. Since "young children mouth and actually eat things that are not food rather indiscriminately" ... "dustfall from airborne lead, if swallowed, can make a significant contribution to a small child's total lead intake and thereby contribute to the occurrence of lead poisoning, especially in urban areas." Id. at VI-20 to 21.

B. The Structure of the Clean Air Act.

14 The Clean Air Act aims to "speed up, expand, and intensify the war against air pollution in the United States with a view to assuring that the air we breathe throughout the Nation is wholesome once again." H.R.REP. No. 91-1146, 91st Cong., 2d Sess. 1 (1970)(emphasis added). To ensure this aim and to prevent the delays that had undercut previous clean air legislation, Congress incorporated health protection procedures that locked the Administrator into protecting people on a timetable. Sections 108-110. To prevent deviations, section 304 authorizes citizens suits to compel the Administrator to perform non-discretionary duties.

15 Section 108(a)(1) triggers the health protection procedures by mandating that certain pollutants* be placed on a "list."

16 The listing of a pollutant requires, inter alia, that the Administrator perform each of the following duties with regard to it:

> Section 108(a)(2) -- the issuance of "air quality criteria" setting forth "all identifiable effects on health and welfare."
>
> Section 109(b)(1) -- the promulgation of "national and primary ambient air standards" which are ceilings on atmospheric concentration which, "allowing an adequate margin of safety, are requisite to protect the public health."
>
> Section 110(a) -- the submission of implementation plans by each state, which may be approved by the Administrator, only if the plan will attain the primary ambient air standard by a statutory deadline.

17 The deadlines of the health protection procedures, taken together, require that health be protected from a pollutant "as expeditiously as practicable" but in no event later than five years and four months after its listing.

C. The Act Compels Action On Lead Under the Health Protection Procedures.

18 The Act required that health be protected from lead no later than May 29, 1976, since section 108(a)(1) required that lead be placed on an initial list of pollutants to be promulgated on January 30, 1971, thirty days after enactment.

19 Section 108(a)(1) states in full:

> "For the purpose of establishing national primary and secondary air quality standards, the Administrator shall within 30 days after the date of enactment of the Clean Air Amendments of 1970, publish, and shall from time to time thereafter revise, a list which includes each air pollutant:

---

* Part IV(C) of this Complaint deals with lead as one of these pollutants.

(A) which in his judgment has an adverse effect on public health and welfare;

(B) the presence of which in the ambient air results from numerous or diverse mobile or stationary sources; and

(C) for which air quality criteria had not been issued before the date of enactment of the Clean Air Amendments of 1970, but for which he plans to issue air quality criteria under this section."

20 In fulfillment of section 108(a)(1)(A), lead has an "adverse effect on public health and welfare" and the Administrator has so found "in his judgment" on numerous occasions and at all relevant times.

21 In fulfillment of section 108(a)(1)(B), lead "results from numerous or diverse mobile or stationary sources" and the Administrator has so found on numerous occasions and at all relevant times.

22 In fulfillment of section 108(a)(1)(C), lead was not the subject of air criteria issued before the date of enactment of the Clean Air Amendments of 1970.

23 Lead, accordingly, meets all the requirements for a pollutant to be listed both at the time of enactment and at all times thereafter.

24 In addition, lead is one of those pollutants "for which [the Administrator] plans to issue air quality criteria under this section" within the meaning of section 108(a)(1)(C). The administration had promised Congress that it intended to treat lead, at the outset, under the health protection procedures* and

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* Statement of Administrator, Environmental Public Health Service, Public Health Service, HEW, Hearings on S. 3229, 3466, 3546, Before the Subcomm. on Air and Water Pollution of the Sen. Comm. on Public Works, 91st Cong., 2d Sess. 196 (1970); Testimony of Administrator, Environmental Public Health Service, Public Health Service, HEW, Hearings on H.R. 12934, 14960, 15137, 15192, 15848, and 15847, Before the Subcomm. on Public Health and Welfare of the House Commerce Comm., 91st Cong., 1st and 2d Sess's. 293 (1970).

Congressional reports state that "the bill would require" such treatment for lead.*

D. The Administrator Has Refused to Act on Lead Under the Health Protection Procedures.

25 The Administrator failed to place lead on the initial list pursuant to section 108(a)(1), has failed to list lead subsequently, and has taken no action to curb lead pollution pursuant to the health protection procedures of sections 108-110.

26 After Plaintiffs brought this failure to the Administrator's attention, he publicly announced his refusal to list lead on December 6, 1973, giving as his sole reason:

> "The determination whether to issue a criteria document for a substance, and thereby set Title I machinery in motion, is discretionary with the Administrator."

27 This interpretation allows the Administrator to sidestep the health protection procedures designed to assure public safety and conflicts with section 108(a)(1)'s mandatory and inclusive tenor: "The Administrator shall publish ... within 30 days ... a list which includes each air pollutant...." (emphasis added).

28 The Administrator put only one pollutant, nitrogen oxides, on the initial list, and in the over three years since then, has not added any other pollutants on the list.

E. Harm to Plaintiffs.

29 The Defendant Administrator's violations of sections 108-110 expose Plaintiffs' members and members of the public generally to unhealthy levels of lead, a toxic air pollutant.

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* S.REP. No. 91-1196, 91st Cong., 2d Sess. 9 (1970). See also H.R.REP. No. 91-1783, 91st Cong., 2d Sess. 44 (1970).

30 Stationary sources of lead, such as smelters and battery factories, create high air lead levels on a localized basis. EPA has, to date, failed to regulate the lead emissions of these sources.

31 Due to this failure, epidemics of lead poisoning have been discovered. Most recently, such an epidemic was discovered around a smelter in Idaho in early September, 1974.

32 Vehicles emit one-half billion pounds of lead from gasoline annually, with the biggest concentrations in urban areas.

33 The Administrator has promulgated, pursuant to section 211, regulations on the lead content of gasoline, which neither can cure his violation of the health protection procedures (§§ 108-110) nor will achieve the aim of protecting health from lead pollution to the extent the hazard comes from lead additives.

34 On January 10, 1973, the Administrator published regulations requiring the use of no-lead gasoline in certain model year 1975 and thereafter vehicles to protect their emission control systems pursuant to section 211(c)(1)(B). 38 Fed. Reg. 1258. But, at the same time, he refused then to issue regulations on the lead content of gasoline to be used in the 100 million pre-1975 vehicles in order to protect health, pursuant to section 211(c)(1)(A).

35 A group of organizations, including some of the Plaintiffs herein, filed a petition for review challenging the delay in the issuance of health protection regulations. NRDC et al. v. EPA, No. 72-2233 (D.C.Cir.).

36 On October 29, 1973, the Court of Appeals ordered EPA to take final action on these regulations within 30 days.

37 On December 6, 1973, the Administrator promulgated fuel additive regulations for lead under section 211(c)(1)(A) which

fall far short of protecting health from this source of lead pollution by May 29, 1976. 38 Fed. Reg. 33734.

38 These regulations, according to EPA, will have no impact on the lead content of gasoline until 1977. Kivel Memo., Nov. 30, 1973.

39 The regulations will leave up to 40% of the lead in gasoline in 1979, the final year of reductions, thereby allowing ambient air lead levels of $2ug/m^3$ in 1979.

40 EPA has never specified an air lead level adequate to protect health, but its last judgment on ambient levels was that $2ug/m^3$ is inadequate to protect health.*

41 Plaintiffs' members and members of the public generally have been exposed to unlawful and unhealthy levels of airborne lead pollution because the Administrator has failed to (a) define a safe air lead level pursuant to section 109(a)(1) and (b) to achieve that level, by any required reduction of emissions from mobile or stationary sources, as expeditiously as practicable but in no case later than May 29, 1976.

F. Special Harm to New York City.

42 The failure of the Administrator to list lead as mandated by section 108(a)(1) of the Act will result in a tripling in the amount of lead contained in the gasoline sold in New York City, starting January 1, 1975.

43 The amount of lead contained in the gasoline sold in New York City has been reduced by steps since 1971. New York City Administrative Code §1403-2-13-11. This reduction caused an

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* EPA's Position on the Health Effects of Airborne Lead IV-5 (Nov. 29, 1972).

immediate reduction in New York City's lead poisoning case load. Letter of March 20, 1973, from New York Commissioner of Air Resources to EPA Administrator.

44 When the federal fuel additive regulations become effective on January 1, 1975, section 211(c)(4) requires that the City's ordinance be preempted.

45 Section 211(c)(4)(C) provides, however, that such preemption will not occur if a non-federal regulation is necessary to achieve a national ambient air standard.

46 If the Administrator had complied with health protection procedures, including the listing of lead and the setting of ambient standards for lead, the City's ordinance would not be preempted since the federal regulations, particularly in the year 1975, are insufficient to achieve any conceivable national ambient air standard throughout New York City.

V. CLAIMS.

Claim I

47 The Administrator has failed to carry out a mandatory duty prescribed by section 108(a)(1) of the Clean Air Act by failing to put lead on the initial list required to be published within 30 days and to list lead at any time thereafter.

Claim II

48 Title 5 U.S.C. §706 provides that:

> "To the extent necessary to decision and when presented, the reviewing court shall decide all relevant questions of law, interpret constitutional and statutory provisions and determine the meaning or applicability of the terms of an agency action. The reviewing court shall
>
> (1) Compel agency action unlawfully withheld or unreasonably delayed."

49 The Administrator has unlawfully withheld and unreasonably delayed agency action which was prescribed by law.

VI. RELIEF.

WHEREFORE, Plaintiffs pray:

(1) That this Court adjudge that the Administrator has violated section 108(a)(1) of the Clean Air Act by his failure to put lead on the initial list of pollutants required to be published by January 30, 1971;

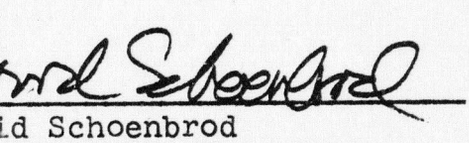
(2) That this Court enter an order compelling the Administrator to implement the health protection procedures of the Clean Air Act (sections 108-110) with regard to lead;

[¶(3) deleted.]

(4) That Plaintiffs be awarded reasonable attorneys' fees and costs of litigation pursuant to section 304 of the Act; and

(5) That Plaintiffs have such other and further relief as this Court deems just and proper.

Respectfully submitted,



David Schoenbrod
Attorney for Plaintiffs
(Natural Resources Defense
Council, Inc.)
15 West 44th Street
New York, New York 10036
(869-0150)

Dated: New York, New York
November 27, 1974

(Original Complaint filed on October 21, 1974)

A 30-A

UNITED STATES DISTRICT COURT

FOR THE SOUTHERN DISTRICT OF NEW YORK

----------X

NATURAL RESOURCES DEFENSE COUNCIL, INC., et al.,

Plaintiffs,

v.

RUSSELL TRAIN, Administrator of the U.S. Environmental Protection Agency, et al.,

Defendants.

CIVIL ACTION No. 74-4617

ORDER

----------X

At the request of Plaintiffs the following allegations of the Amended Complaint dated November 27, 1974, are hereby striken to correct clerical errors:

(a) page 2 - the sentence beginning with the words "The Administrator's refusal to perform...." and ending with the words "....Order of March 8, 1974).";

(b) page 2 - the clause beginning with the words "....and, accordingly, to prevent...." and ending with the words "....on January 1, 1975."; and

(c) pages 13-14 - paragraphs 42, 43, 44, 45, and 46 in their entirety.

SO ORDERED.

United States District Court Judge

protection procedures of the Clean Air Act. Sections 108-110*. These procedures require the Administrator to promulgate a national primary ambient air standard for lead, requisite to protect health, and to attain this standard no later than May 29, 1976.

Plaintiffs, organizations concerned with children's health and the environment, bring this action under the Citizens Suit Section of the Clean Air Act, to compel the treatment of lead under the Act's health protection procedures.

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* The Clean Air Act is codified as 42 U.S.C. §1857 et seq. The sections of the Act relied upon herein are:

Section 108	42 U.S.C. §1857(c)(3)
Section 109	42 U.S.C. §1857(c)(4)
Section 110	42 U.S.C. §1857(c)(5)
Section 211	42 U.S.C. §1857f-6c
Section 304	42 U.S.C. §1857h-2

Since the code citation becomes confusing, especially when subsections and subparagraphs are cited, this Complaint hereinafter will cite to the Act.

BEST COPY AVAILABLE

fall far short of protecting health from this source of lead pollution by May 29, 1976. 38 Fed. Reg. 33734.

38 These regulations, according to EPA, will have no impact on the lead content of gasoline until 1977. Kivel Memo., Nov. 30, 1973.

39 The regulations will leave up to 40% of the lead in gasoline in 1979, the final year of reductions, thereby allowing ambient air lead levels of 2ug/m^3 in 1979.

40 EPA has never specified an air lead level adequate to protect health, but its last judgment on ambient levels was that 2ug/m^3 is inadequate to protect health.*

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[¶¶42-46 deleted.]

---

* EPA's Position on the Health Effects of Airborne Lead IV-5 (Nov. 29, 1972).

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>
> (1) Compel agency action unlawfully withheld or unreasonably delayed."

A 31

UNITED STATES DISTRICT COURT

FOR THE SOUTHERN DISTRICT OF NEW YORK

NATURAL RESOURCES DEFENSE COUNCIL, INC., et al., Plaintiffs, v. RUSSELL TRAIN, Administrator of the U.S. Environmental Protection Agency, et al., Defendants.	NOTICE OF MOTION FOR SUMMARY JUDGMENT, DECLARATORY JUDGMENT, AND MANDAMUS
	74 Civ. 4617

S I R S :

PLEASE TAKE NOTICE that the Plaintiffs, Natural Resources Defense Council, Inc. ["NRDC"] et al., upon the annexed Statement of Material Facts, Memorandum of Law, and affidavits of Dr. Roger Challop (Nov. 18, 1974), Dr. Oliver David (Nov. 14, 1974), Dr. Laurence Finberg (Nov. 20, 1974), David Schoenbrod (Nov. 25, 1974), William Shapiro (Nov. 12, 1974), and Marcia Tompkins (Nov. 21, 1974), all affidavits being sworn to on the dates indicated, will move this Court on the 16th day of December, 1974, at 4:00 P.M., or as soon thereafter as counsel can be heard, in Room 1305 of the United States Court House, Foley Square, New York, New York for an order (1) granting summary judgment in favor of Plaintiffs pursuant to Rule 56 of the Federal Rules of Civil Procedure, (2) declaring that the Defendants have violated section 108(a)(1) of the Clean Air Act (42 U.S.C. §1857c-3(a)(1)) by their failure to place lead on a list of pollutants to be issued within 30 days from enactment of the Clean Air Act, pursuant to Rule 57 of the Federal Rules of Civil Procedure and 28 U.S.C. §§2201, 2202, and (3) compelling the Defendants to implement

sections 108-110 of the Clean Air Act (42 U.S.C. §1857c-3-4-5) with regard to lead, pursuant to 42 U.S.C. §1857h-2 and 28 U.S.C. §1361.

PLEASE TAKE FURTHER NOTICE that Defendants answering papers will be served by hand no later than December 9, 1974, and that Plaintiffs' reply papers will be served by hand no later than December 12, 1974.

PLEASE TAKE FURTHER NOTICE that this motion will be submitted at the above designated time and place unless attorneys for Defendants or this Court request oral argument.

November 26, 1974

David Schoenbrod
Attorney for Plaintiffs
(Natural Resources
Council, Inc.)
15 West 44th Street
New York, New York 10036
(212) 869-0150

TO: PAUL CURRAN, ESQ.
United States Attorney
Attorney for Defendants
U.S. Court House
Foley Square
New York, New York 10007

JOSEPH McLAUGHLIN
Shearman & Sterling
Attorney for Exxon Corp.
53 Wall Street
New York, New York 10005

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Supporting Papers

Table of Contents

STATEMENT OF MATERIAL FACTS — Attached Separately

MEMORANDUM OF LAW — Attached Separately

AFFIDAVITS:

DR. ROGER CHALLOP

DR. OLIVER DAVID

DR. LAURENCE FINBERG

DAVID SCHOENBROD
- Appendix A: Memorandum of Nov. 30, 1973, from Joseph Kivel to Victor Krimm
- Appendix B: Letter of March 20, 1973, from Fred C. Hart to William Ruckelshaus
- Appendix C: Memo of April 11, 1972, from Gerald Hansler to John T. Middleton
- Appendix D: "Idaho Department of Health and Welfare, Survey of Heavy Metals Contamination, Shoshone County, Status Report, Oct. 2, 1974."

WILLIAM SHAPIRO

MARCIA TOMPKINS
- Exhibit A: EPA Order No. 1130.8, order reconstituting National Air Quality Criteria Advisory Committee
- Exhibit B: Committee Membership List
- Exhibit C: Committee Agenda, Nov. 14, 1974, Meeting
- Exhibit D: Memo to the Committee from B.J. Steigerwald
- Exhibit E: Submission to the Committee from Natural Resources Defense Council, Inc.

UNITED STATES DISTRICT COURT
FOR THE SOUTHERN DISTRICT OF NEW YORK

NATURAL RESOURCES DEFENSE COUNCIL, INC., et al., Plaintiffs, v. RUSSELL TRAIN, Administrator, U.S. ENVIRONMENTAL PROTECTION AGENCY, et al., Defendants.	CIVIL ACTION NO. 74-4617 AFFIDAVIT

STATE OF NEW YORK)
: ss.:
COUNTY OF NEW YORK)

ROGER S. CHALLOP, being duly sworn, deposes and says:

1. I am a Doctor of Medicine, duly licensed to practice medicine in the State of New York. I am an Associate in Pediatrics and Attending Physician at Babies Hospital and St. Luke's Hospital and an Associate Director of the Pica Clinic at the Columbia Presbyterian Medical Center, all in New York City. I maintain an office for the practice of Community Medicine at 615 West 164 Street, New York, New York.

2. I graduated from Brooklyn College in 1963 with the degree of Bachelor of Science in Psychology. I received my M.D. degree from the New York Medical College in 1967.

3. I was an intern at the Brookdale Hospital Center from 1967 to 1968 and a resident in Pediatrics at the Mount Sinai Medical Center, New York, New York, from 1968 to 1969. I was the Chief Resident at Mount Sinai Medical Center from 1969 to 1970. I served on active military service from 1970 to 1972 as Pediatric Consultant (Surgeon - Lt. Commander), working

with the United States Public Health Service in the Bureau of Community Environmental Management's Childhood Lead Poisoning and Accident Prevention Programs in Cincinnati, Ohio. I have also served since 1971 as an Instructor in Pediatrics at both the Children's Hospital, University of Cincinnati College of Medicine, and at the College of Physicians and Surgeons, Columbia University. I am, and have been since 1970, Pediatric Consultant to the United States Public Health Service. Since 1970, I have also served as Pediatrician at Batavia Children's Clinic. I was licensed by the National Medical Board in 1968. I am a Diplomate of the American Board of Pediatrics, having passed the examination in April of 1974.

4. I am a member of the Medical Advisory Committee of the New York Scientists' Committee for Public Information and the Lead Poisoning Committee of the Public Interest Campaign, Inc. I am and/or have been a member of the National Association of Interns and Residents; Phi Chi Medical Fraternity; American Medical Association; Bela Schick Society, Mount Sinai Hospital; and a National Member of Smithsonian Associates.

5. I hold and/or have held the following appointments: Liaison Member, American Academy of Pediatrics (Committee on Accident Prevention) 1970 to present; Chairman, United States Public Health Service Task Force on Childhood Lead Poisoning, 1970; Member, Department of Health, Education and Welfare Intradepartmental Committee on Childhood Lead Poisoning, 1970-1972; Member, Ad Hoc Committee on the Daily Permissible Intake of Lead in Children, 1971; Liaison Representative, Subcommittee on Medical and Laboratory Issues, Department of Health, Education and Welfare Intradepartmental Committee for Planning and Implementation of the Lead-Based Paint Poisoning Prevention Act

(PL 91-695), 1972; Member, Subcommittee on Prevention of Burn Injuries, American Academy of Pediatrics Committee on Accident Prevention, 1972; Member, Technical Review Committee for Community Lead Poisoning Grants under PL 91-695, 1972; Contract Officer and Consultant, Lead Poisoning Contracts involving pica, dose and time-related effects of lead and other trace metals, out-patient therapy, microblood and body lead burden analysis, educational materials and an abstract review of literature on lead and trace metal intoxication in children, 1971-1972.

6. I received the Superior Performance Award from the United States Department of Health, Education and Welfare in 1972 for my work in developing and organizing the national effort to prevent lead-based paint poisoning in children.

7. I am the author or co-author of 10 publications on the health problems associated with lead and have participated in eight workshops in this field. I have also served as technical consultant or contributed to 15 additional publications on health problems associated with lead.

8. I am familiar with most of the published literature and research with respect to the physiological effects of airborne lead and in particular with such effects as they relate to infants and children. I am thoroughly familiar with the on-going studies and assessments of reduced lead levels in gasoline by the U.S. Environmental Protection Agency, as well as New York City Environmental Protection Administration's experience with its lead in gasoline standards. I am familiar with the New York City experience of reduced lead in street dirt and a concomitant decrease in the reported incidents of childhood lead poisoning since the time these standards went into effect. It must be added, however, that a vigorous childhood lead screening

program was being carried out by the New York City Health Department at the same time.

9. I am thoroughly familiar with the dustfall lead theory (that atmospheric fallout of lead is the primary source of measured lead content of urban street dust) and it is my medical opinion that dustfall lead represents a significant health hazard in New York City, in particular with respect to infants and children. The retention of lead by the body is cumulative and adds to the total body burden from whatever source. For that reason and inasmuch as there is no known biological value in lead and its potential toxicity has been demonstrated, it is medically advantageous to reduce human exposure to lead from all sources to the lowest possible level, particularly among infants and children in an urban environment. Recent evidence about elevated blood lead levels (40 to 59 micrograms per 100 milliliters whole blood) that were below levels set for active drug chelation therapy (60 micrograms per 100 milliliters whole blood) has raised concern about the large number of urban children in this category. In an epidemologic follow-up study of an El Paso, Texas, smelter and its fallout radius, investigators found that children who had had blood lead levels about 40 micrograms at the time of the original study done in 1972, as compared with a control group of matched children who were below that level at the same time, showed a significant reduction in motor performance IQ (but not verbal IQ) as compared to the control. The authors concluded, "This study suggests that children with blood lead levels in the 40 to 80 micrograms per 100 milliliter range may display subclinical impairments in a broad range of psychological function." (Center for Disease control Morbidity and Mortality Report, Vol. 23, No. 18, May 4, 1974).

10. While it is medically helpful to reduce all exposure to lead from all sources (lead based paint, lead dustfall, etc.) in children and adults, it is especially crucial in those first three years of age, because of lowered physical protection by a lack of shielding of the brain in this age group. Higher than expected (20 to 30 micrograms per 100 milliliters whole blood) umbilical blood lead levels in some urban newborns, which could only have come from maternal transfer, have been reported. As long as the newborn starts out with a birthright of a greater than zero lead level, he or she is virtually more at risk to reach unsafe levels during childhood if exposure continues. It is because of this desire to reduce the baseline lead exposure of children that I respectfully submit that the current New York City ordinance not be preempted by the federal standards.

ROGER S. CHALLOP

Sworn to before me this
18th day of November, 1974

CAROL ANN HINE
NOTARY PUBLIC, STATE OF NEW YORK
NO. 31-4512712
QUALIFIED IN NEW YORK COUNTY
COMMISSION EXPIRES MARCH 30, 1975

UNITED STATES DISTRICT COURT
FOR THE SOUTHERN DISTRICT OF NEW YORK

NATURAL RESOURCES DEFENSE COUNCIL, INC., et al.,

Plaintiffs,

v.

RUSSELL TRAIN, Administrator, U.S. Environmental Protection Agency, et al.,

Defendants.

CIVIL ACTION NO. 74-4617

AFFIDAVIT

STATE OF NEW YORK)
COUNTY OF KINGS) ss:

OLIVER J. DAVID, being duly sworn, deposes and says:

1. I am a Doctor of Medicine, duly licensed to practice medicine in the States of New York and California, an assistant professor of psychiatry at the Downstate Medical Center of the State University of New York ("Downstate Medical Center"), and maintain an office at 450 Clarkson Avenue, Brooklyn, New York.

2. I received a Bachelor of Science degree from the City College of New York in 1958, an M.D. degree from Downstate Medical Center in 1962, and a Doctor of Medical Science degree from Downstate Medical Center in 1971.

3. I served my internship at Mt. Zion Hospital, San Francisco, California in 1962-63, was a captain in the United States Air Force Medical Corps in 1963-65. I was a resident in psychiatry at Hillside Hospital, Queens, New York in 1966-67 and at Kings County Hospital, Brooklyn, New York in 1967-69.

I have been affiliated with Downstate Medical Center as an instructor in psychiatry in 1968-70, as a clinical assistant professor of psychiatry in 1970-73 and as an assistant professor of psychiatry since early 1973. I have also served as a consulting psychiatrist for the South Beach Psychiatric Center of the New York State Department of Mental Hygiene from 1971 to the present time.

4. I was certified by the National Board of Medical Examiners in 1963 and by the Board of Psychiatry and Neurology in 1971.

5. I have concentrated my research at Downstate Medical Center on the relationship in children between lead concentrations in the blood and urine and hyperactivity.

6. I am the primary author of an article describing this work through 1972, entitled "Lead and Hyperactivity," which appeared in the October, 1972 issue of the medical journal Lancet. Very high cencentrations of lead are known to cause massive brain damage in children. It is logical that lesser concentrations of lead would cause lesser degrees of brain damage, although no direct experimental proof is available at this time. The best evidence now obtainable of the effects of lead assimilation on children's brain tissue is the correlation of blood concentrations of lead with clinically observable manifestations of brain dysfunction.

7. Hyperactivity is the most common sympton of the syndrome termed "minimal cerebral dysfunction" or, in lay language, minimal brain damage. The research referred to in paragraph 6 involved the determination of the concentration of lead in the

blood and urine of approximately 60 children who had been determined to be hyperactive and approximately 35 children (the control group) who had been determined to be not hyperactive. Blood lead levels of 20 micrograms ("ug") per 100 milliliters ("ml") are generally regarded by medical experts as "safe" and 60 ug per 100 ml levels are almost universally considered to be toxic, with no consensus at present as to the range between. The mean blood concentrations of lead among the hyperactive children was 26 ug per 100 ml compared with 22 ug per 100 ml among the control group. More startling, approximately 50 percent of the hyperactive children had a blood concentration of lead between 20 ug per 100 ml and 60 ug per 100 ml, while by contrast, only 15 percent of the control group had a blood concentration in that range. The research, therefore, indicated a positive correlation in children between elevated blood concentrations of lead and hyperactivity, and strongly suggests that the assimilation of lead by children may be a cause of hyperactivity. On the basis of this work, I am recommending a re-evaluation by the medical profession of the blood concentrations of lead in children which should properly be regarded as "safe."

8. Since the research described in paragraphs 6 and 7, I have continued to concentrate my work on the relationship between low-level lead exposure and brain dysfunction in children. This research both corroborates the initial research and further suggests that, in some children, low-level lead exposure may be causal of minimal brain damage. This additional research consists of two studies described in paragraphs 9 and 10.

13. To the extent that the assimilation of such lead from the environment may be a cause, alone or in combination with other causes, of hyperactivity leading to problems in education or possibly a cause of more serious dysfunctions, it is my opinion that lead in the environment is a proper subject of public concern and should be reduced to the greatest extent possible.

OLIVER J. DAVID

CAROL ANN HINE
NOTARY PUBLIC, STATE OF NEW YORK
NO. 31-4512742
QUALIFIED IN NEW YORK COUNTY
COMMISSION EXPIRES MARCH 30, 1975

Sworn to before me this
14Th day of November, 1974

Carol Ann Hine

UNITED STATES DISTRICT COURT
FOR THE SOUTHERN DISTRICT OF NEW YORK

NATURAL RESOURCES DEFENSE COUNCIL, INC., et al., Plaintiffs,	:	
v.	:	
RUSSELL TRAIN, Administrator, U.S. Environmental Protection Agency, et al., Defendants.	:	CIVIL ACTION NO. 74-4617
	:	AFFIDAVIT

STATE OF NEW YORK)
ss:
COUNTY OF BRONX)

LAURENCE FINBERG, being duly sworn, deposes and says:

1. I am a Doctor of Medicine, duly licensed to practice medicine in the State of New York and am, and have been since 1963, Chief of the Department of Pediatrics at the Montefiore Hospital and Medical Center, Bronx, New York, and Attending Pediatrician at Bronx Municipal Hospital Center.

2. I am a graduate of the University of Chicago, where I received a Bachelor of Science degree in Physiology in 1944 and a Doctor of Medicine degree in 1946. I was an intern at the University of Chicago Clinics from 1946 to 1947 and served on active military service as Senior Assistant Surgeon, United States Public Health Service (Venereal Disease Division, Rapid Treatment Center, Hot Springs, Arkansas) from 1947 to 1949. I was an Assistant Resident and Resident in Pediatrics at the Baltimore City Hospitals from 1949 to 1951. From 1951 to 1963, I served continuously at the Baltimore City Hospitals, holding the following positions: Assistant Chief Pediatrician, 1951-1961; Director of Pediatric Outpatient Department, 1951 to 1963; Director of Premature Nursery, 1951-1959; Associate Chief of Pediatrics, 1961-

1963. I also served as Pediatrician for the Harriet Lane Home from 1951 to 1963. I was certified by the American Board of Pediatrics in 1952.

3. I was an instructor in the Department of Pediatrics, Johns Hopkins University School of Medicine from 1951 to 1956, and Assistant Professor of Pediatrics at that institution from 1956 to 1963. I am, and have been since 1963, Professor of Pediatrics at the Albert Einstein College of Medicine, New York City. I am a member of Phi Beta Kappa, Alpha Omega Alpha and the Society of the Sigma Xi. I served as Editor of the Section on Nutrition, Barnett's Pediatrics 15th Edition, published in 1971.

4. I was the Consultant Pediatrician at the Sinai Hospital of Baltimore from 1959 to 1963. From 1964 to date, I have been Consultant in Pediatrics at Jewish Memorial Hospital, New York City, and from 1965 to date, I have been Pediatric Consultant to the United States Children's Bureau. I am, and have been since 1969, an Examiner for the American Board of Pediatrics. From 1970 to date, I have served as Consultant Pediatrician for Blythedale Children's Hospital, Valhalla, New York, and Phelps Memorial Hospital, Tarrytown, New York.

5. I am and/or have been a member of the following societies and committees: American Pediatric Society; Society of Pediatric Research; American Academy of Pediatrics (Committee on Environmental Hazards, 1968 to date); Association of Medical School Pediatric Department Chairmen, Inc.; American Institute of Nutrition; Association for Pediatric Ambulatory Services; American Society for Clinical Nutrition; American Federation for Clinical Research; American Society of Nephrology; International Society of Nephrology; American Association for the Advancement of

Science; American Medical Association; National Society for Medical Research (representing academic pediatric societies); Pan American Medical Association.

6. I am also a member of the following societies and committees: Bronx Pediatric Society (President, 1969-1970); the Harvey Society; Medical Society of the State of New York (Bronx County); New York Academy of Medicine (Chairman of the Pediatrics Section, 1967); New York Academy of Science; New York Diabetes Association; Pediatric Advisory Committee of the New York City Department of Health (1970 to date).

7. I am the author or co-author of more than 70 published articles in medical science, principally related to pediatrics, including an article in collaboration with H. Roffman entitled "Lead Poisoning in Children - A Disease of the Environment," published in Health News by the New York State Department of Health in July, 1969.

8. I am familiar with most of the published literature and research with respect to the physiological effects of airborne lead and in particular with such effects as they relate to infants and children.

9. I am familiar with the dustfall lead theory (that atmospheric fallout of lead is the primary source of measured lead content of urban street dust) and it is my medical opinion that dustfall lead represents a significant health hazard in New York City, in particular with respect to infants and children. From my own clinical experience, there are urban children who were dirt eaters with elevated blood leads and signs of toxicity where painted surfaces in the household and other parts of their environment could not be incriminated.

10. There is no known biological value in lead and its potential toxicity has been documented. Inasmuch as the retention of lead by the body is cumulative and, by adding to the overall body burden, may aggravate an already compromised system, any reduction in the lead content of the urban atmosphere from whatever source would be medically helpful, particularly to the extent that such reduction would contribute to a corresponding reduction in the lead levels in street dust. Correspondingly, an increase in the lead content of the urban atmosphere presents a danger by increasing the retention of lead.

11. The effects of excessive exposure to lead are not limited to the period of exposure since the body accumulates lead and since lead may cause life-long medical problems.

LAURENCE FINBERG

Sworn to before me this
20th day of November, 1974

Eva Morfi

EVA MORFI
COMMISSIONER OF DEEDS
CITY OF NEW YORK NO. 3-424
Certified in Bronx County
Commission Expires March 30, 1976

UNITED STATES DISTRICT COURT

FOR THE SOUTHERN DISTRICT OF NEW YORK

- x

NATURAL RESOURCES DEFENSE COUNCIL, INC., et al.,

Plaintiff,

v.

RUSSELL TRAIN, as Administrator of the U. S. Environmental Protection Agency,

Defendants.

AFFIDAVIT

74 Civ. 4617

- x

STATE OF NEW YORK)
: ss.:
COUNTY OF NEW YORK)

DAVID SCHOENBROD, being duly sworn, deposes and says:

1. I am attorney for Plaintiffs in this action and am employed as a staff attorney at the Natural Resources Defense Council, Inc.

2. Attached to this affidavit are copies of documents in the files of the Environmental Protection Agency made from the Appendix filed in the case of Natural Resources Defense Council v. EPA (D. C. 74-1023).

Appendix A: Memorandum of Nov. 30, 1973 from Joseph Kivel to Victor Krimm.

Appendix B: Letter of March 20, 1973 from NYC Commission of Air Resources' Fred C. Hart to EPA Administrator William Ruckelshaus, together with Appendix C to the letter.

Appendix C: Memorandum of April 11, 1972 from EPA Regional Administrator Gerald M. Hansler to EPA Deputy Assistant Administrator John T. Middleton.

APPENDIX A

BEST COPY AVAILABLE

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3. Attached as Appendix D to this affidavit is a document entitled "Idaho Department of Health and Welfare, Survey of Heavy Metals Contamination, Shoshone County, Status Report, Oct. 2, 1974." I received this report from a private citizen in Idaho. I telephoned the Idaho Department of Health and Welfare and verified, with Mr. Anthony Yankel, Environmertal Engineer, that this document was an accurate copy of the report issued by the Department.

David Schoenbrod

DAVID SCHOENBROD

Sworn to me this 25th day of November, 1974

Carol Ann Hine

CAROL ANN HINE
NOTARY PUBLIC, STATE OF NEW YORK
NO. 31-4512742
QUALIFIED IN NEW YORK COUNTY
COMMISSION EXPIRES MARCH 30, 1975

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY

A 49

Rec. 0559

SUBJECT: Final Leaded Gasoline Regulations

DATE: Nov. 30, 1975

FROM: Joseph Kivel

TO: Victor Kimm, Acting DAA
for P&E

A comparison of the final regulations and proposed regulations are presented in the tables below.

Total Pool Averaging

Year	Proposed Regulations	Final Regulations
1975	1.4 g/gal	1.7 g/gal
1976	1.1	1.4
1977	0.8	1.0
1978	0.6	0.8
1979	--	0.5

Leaded Gasoline Only*
(1973 Average = 2.25 g/gal)

Year	Proposed Regulations	Final Regulations**
1975	2.0	2.36
1976	1.7	2.26
1977	1.5	1.89
1978	1.25	1.76
1979	--	1.25

*Calculations assumed all post-1974 autos will use lead-free gasoline.

**"Actual" values will be somewhat lower since not all 1975 autos will use catalysts.

Conculsion

In effect, these regulations require no reduction in lead content of leaded gasoline in 1975 and 1976; a reduction of half that originally proposed by 1978; and a reduction to 1.25 g/gal by 1979 rather than 1978. A reasonable compromise!

APPENDIX B

EPA Form 1320-6 (Rev. 6-72)

A 50 Fed. Admin App. 262

DEPARTMENT OF AIR RESOURCES
51 Astor Place, New York, N.Y. 10003

FRED C. HART, COMMISSIONER

Rec. 1256

March 20, 1973

SPECIAL DELIVERY

Mr. William J. Ruckelshaus
Administrator
Environmental Protection Agency
Waterside Mall
Fourth and "M" Streets, S.W.
Washington, D.C. 20036

Dear Mr. Ruckelshaus:

I am writing in response to your request for comments on the reproposed regulations providing for a reduction in the lead content of leaded gasoline for health protective reasons. 38 Fed. Reg. 1258 et seq.

In my opinion, the reproposed regulations fail to incorporate your agency's position on the health effects of ambient lead and are inadequate to protect the health of the citizens of New York City.

At present, while 75 percent of the gasoline consumed in this City is purchased here and thus contains only 1 gram of lead per gallon, our high traffic areas continue to have ambient lead readings of 5 micrograms per cubic meter on about 15 percent of all 24 hour sampling periods. These lead concentrations are unhealthy of themselves and further represent over 14 percent of the federal particulate health standard less background particulates.

For this reason I urge you to either (i) promulgate stricter standards at least for areas like New York City with demonstrable health problems; (ii) permit areas with demonstrable health problems to enforce stricter local regulations; or (iii) propose and promulgate air quality criteria and ambient standards for lead.

As far as the New York City program is concerned, we have limited the lead content in gasoline since November 1, 1971. The relevant standards are:

1) On or after November 1st, 1971	2.0 gram per gal.	1.5 gram per gal.
2) On or after January 1st, 1972	1.0 gram per gal.	1.0 gram per gal.

APPENDIX C

Prior to November 1, 1971, it is assumed that lead content in gasoline sold in New York City was similar to gasoline sold in the rest of the region and that measurements of lead in gasoline outside New York City in 1972 would reflect the lead in gasoline in New York City prior to November 1, 1971.

Appendix A indicates that within New York City during November, 1972 and February, 1973, lead levels in gasolines sampled averaged 0.91-0.93 grams per gallon. During January, 1973, lead levels outside New York City, but within the Region, in gasolines sampled, averaged 2.51 grams per gallon for premium gasoline and 1.93 grams per gallon for regular gasoline.

Appendix B indicates a 37% decrease in ambient lead levels due to the change in lead levels in gasoline within New York City even based on 25% of the gasoline that is used in the City is purchased outside the City.

Appendix C is a statement by our technical staff as to the health and other effects of lead.

Sincerely,

Fred C. Hart by JK.

Fred C. Hart, P. E.
Commissioner.

encs,

FCH/jb

The soil and our water supplies are a constant sink for atmospheric lead, which for 90% originates from motor vehicle exhaust, i.e., from lead additives to gasoline. Airborne lead appears as an aerosol and is also associated with carbon particles. Lead alkyls are volatile and fat soluble, hence their inherent danger to the central nervous system (CNS). Lead not only builds up in the soil and its produce but also in water bodies, where it is deposited on the bottom; hence its accumulation in oysters and other shelfish. Thus lead in the atmosphere poisons man via the food chain, via our drinking water and via direct inhalation into the respiratory tract. In the human body and especially in young children, lead affects enzymes and hame synthesis, and as mentioned above, it affects the CNS. Accumulating in bone and kidney, it has potential long term effects. According to the New York City Bureau of Lead poisoning Control, its case load peaks in summer, a fact which may be substantiated by much poorer atmospheric ventilation in that season and by the fact that small children in summer pass most of the day outdoors so that they are more directly exposed to airborne automative lead. The Bureau also indicates that its case load has dropped appreciably since November 1971, when New York City ordered the gasoline sold within its limits, to contain less lead addition than that sold in neighboring counties.

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A recent study has shown lead levels of gasoline sold in Nassau and Westchester counties to be appreciably higher than those in the City (Average 2.51 g/gallon for premium and 1.93 g/gallon for regular against 1/2g/gallon). Consequently, more lead is brought into New York City air by commuters than by New York residents.

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ENVIRONMENTAL PROTECTION AGENCY

Rec. 0183
App. 0349

Date: APR 11 1972

Reply to Attn. of: Gerald M. Hansler, P.E.
Regional Administrator

Subject: Proposed Regulations of Fuels and Fuel Additives (proposed 40 CFR Part 80)

To: Dr. John T. Middleton
Deputy Assistant Administrator for Air Programs
Parklawn Bldg.
5600 Fishers Lane, Rm. 17-59
Rockville, Maryland 20852

The Region II Office has solicited comments from State and local agencies and received the following responses:

1. N. J. State Department of Environmental Protection, City of Newark Department of Health, Niagara County Health Department and Westchester County Air Pollution Agency concur in the regulations.

2. Suburban N. J. Air Pollution Agency and Erie County Health Department state that they have nothing to add.

3. N. Y. State Department of Environmental Conservation requests that a quicker time table for lead phase-out be created. Low lead fuels are a requirement for New York State's proposed NO_x retrofit control program in New York City for gasoline powered trucks. Lead deposits plug up the recycle gases required to control NO_x formation. Furthermore, lower lead in gasoline will reduce vehicle particulate emissions and help enable New York City to meet primary particulate standards. Finally, New York City currently is exceeding the recommended ambient air lead level of 2 ug/m^3 three month average and needs relief as soon as possible.

4. N. Y. City Department of Air Resources points out that New York City has three month average lead readings of 28 ug/m^3 at its Queens-Midtown Tunnel toll booths and three month geometric readings of 5.19 ug/m^3 on the Queens Borough Hall roof. The New York City agency believes that the regulations will not bring the ambient air lead to safe levels. It protests that except to meet particulate levels required by the implementation plan, New York City will not be able to

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deal directly with the lead content in gasoline to combat the health endangerment presented by lead per se. New York City demands regulations with a more complete removal of lead in gasoline, and at a quicker rate, than the regulations now proposed.

This office believes that the regulations are reasonable in light of the evidence presented in A.P. Altshuller's "Effects of Reduced Use of Lead in Gasoline on Vehicle Emissions and Photochemical Reactivity." The proposed regulations should bring lead levels to 2 ug/m^3, three month average, and at the same time insure that no additional environmental degradation will occur from polynuclear aromatic (PNA) emissions.

In answer to New York State's concern over the regulations, this office encouraged New York to attend the upcoming hearings or submit testimony to the Administrator. It was also suggested that the New York City trucks be retrofitted for NO_x control so as to be able to use lead free fuels, which will be widely available in 1974.

However, it should be recognized that the concentrates experienced in New York City constitute a serious health hazard at present. Therefore, I recommend that New York City be permitted to adopt more stringent regulations than those proposed.

G. M. Hansler

Gerald M. Hansler, P.E.
Regional Administrator

IDAHO DEPARTMENT OF HEALTH AND WELFARE

A 56

SURVEY OF HEAVY METALS CONTAMINATION
SHOSHONE COUNTY, IDAHO
STATUS REPORT
October 2, 1974

I. BACKGROUND

Through a routine monitoring program, the Department of Health and Welfare became aware of increasing levels of lead in the ambient air in the vicinity of Kellogg, Idaho, from 1971 to 1973. Although no specific numerical air quality standards exist for lead and no health problem attributable to the lead concentrations had been reported, the Department concluded that a special study of the situation was warranted. It was felt that the source and the ultimate fate of the lead should be determined. Shortly after a limited environmental study was initiated by the Department in early 1974, two cases of lead absorption in children from Kellogg were reported. In response, the environmental survey of heavy metals in the area was intensified and carried out in conjunction with a comprehensive study of human lead absorption in Shoshone County. While lead was the primary subject of the survey, other heavy metals were also of concern. The environmental studies were outlined as follows:

A. Hi-vol air sampling in population centers. Twenty four hour samples will be collected every other day in order that reasonably accurate monthly averages and variations may be calculated. Air sampling will be done at the following distances from the smelter; less than 1 mile, 1.5 miles, 2 miles, 3 miles, 7 miles, 11 miles and 17 miles.

B. Soil samples will be collected in three phases: 1) an intensive sampling survey will be made during the first 10 days of June when approximately 150 samples will be taken at general sites (school yards, playgrounds, residential yards) within population centers from less than 1 mile to 17 miles from the smelter. 2) an extensive sampling survey will be made during the latter part of June and July when samples will be taken from the entire valley using an appropriate sampling grid. 3) a specific sampling survey will be done in conjunction with the human (blood sample) survey at which time samples will be taken from the yards of the children selected for blood sampling.

C. Vegetation samples will be taken in the same manner as described for soil sampling. Vegetation taken during the human survey will include garden vegetable samples.

D. Water samples will be taken from the municipal water systems and from other sources of water used for human and animal consumption.

E. House dust samples will be taken at each surveyed residence during the human survey and from schools and other public buildings.

This report is a summary and preliminary analysis of data collected in Phase I of the environmental survey, along with a very limited presentation of available data from Phase III.

II. PHASE I REPORT:

In Phase I of the study, the purpose was to find where lead was present in the general environment and what possible sources of lead contamination have contributed, or continue to contribute, to the problem. It must be remembered that in this phase the specific home environment is not addressed, but the study is designed to observe the general environment, (air, water, soil and vegetation.)

Significant and abnormal levels of lead in each of these categories were found throughout the valley. Levels of lead higher than those found in the existing literature are consistently observed in the Kellogg-Smelterville area. Several of the samples were screened for other heavy metals (zinc, cadmium, arsenic, mercury and antimony.) It is suspected that the cadmium levels observed may constitute a human health hazard in some areas. Antimony and zinc are present in significant amounts in many areas; however, the health effects of the environmental contamination of these metals is not known. A local veterinarian has cited many animal deaths in the area as caused by antimony and cadmium, as well as lead poisoning.

Because of the immediacy of the problem, attention has been focused on lead. In comparison to published information, the lead levels in the communities of Kellogg and Smelterville are as high, or higher, than any recorded elsewhere.

As this summary is read, it must be remembered that the data set here is incomplete. There are several observations and parameters regarding each site that are not included. What follows in a nominal analysis and should be reviewed as such. At some time in the near future, a rigorous analysis will be performed that will include analysis of all variables and all appropriate parameters. At that time, some relationships may become evident that are too subtle to surface in this cursory approach.

A. Air

Data on the concentrations of lead in the ambient air exist from 1971 to the present. Until May of this year that data consisted of three stations. These stations are denoted by a star in the following table.

STATION NAME	LOCATION	DISTANCE FROM SMELTER
Silver King	Silver King School, Smelterville	.3 mi
*Smelterville	City Hall, Smelterville	.9 mi.
*Kellogg Medical Center	Kellogg Doctors Clinic, Kellogg	1.8 mi.
*Kellogg	City Hall, Kellogg	2.0 mi.
Pinehurst	Elementary School, Pinehurst	3.5 mi.
Osburn	Radio Station, Osburn	7.9 mi.
Wallace	Post Office, Wallace	11.8 mi.
Mullan	Elementary School, Mullan	18.5 mi.
Cataldo	Commercial Building, Cataldo	7.9 mi.

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In conjunction with this study, the six additional high volume samplers listed above were added to the network. We would consider readings in excess of a 1.5 ug/m^3 to be significantly above normal readings for rural areas of this character. Abnormal readings have been observed on all but the Mullan monitor, however, abnormal lead concentrations are uncommon in Wallace and Cataldo. Consistent above normal readings of lead in the ambient air were found from Osburn to Kingston. The suspected sources of air contaminant in this area are as follows:

Suspected Source	Concentration of Pb	Volume	Effect Area
Area Sources			
Automobiles	100 ug/m^3 in exhaust	~ 1T/mo	~10^2 ft. roads
Soil & dust	0-2%		Entire valley
Ore & concentrate transportation	5-60%		~ 10^2 ft. railroads
Point Sources			
Bunker Hill Smelter Blast Furnace & Main Stack	50-70%	~20-100 T/Mo	Entire Valley
Bunker Hill Fugitive Sources	20-70%	~10-20 T/Mo	< 10 miles
Milling Operations	5-15%		< 1 mile

Ambient air concentrations of lead have been increasing regularly since the beginning of our records. This increase can be seen in Figure 1 (lower plot). The upper plot in Figure I represents the readings obtained for total suspended particulate. As the ambient lead and particulate concentrations do not increase proportionately, an increase in percent lead in the particulate matter has been evident. This trend is shown in Figure 2. These observed percentages of lead in the particulate matter are much higher than any observed in the soil thus far. This would suggest that the bulk of the lead in the ambient air originates from those sources with higher concentrations of lead. Those sources are concentrate and smelting activities.

Two months of ambient air data from the high volume sampler network in the valley have been completed and analyzed. Those months are June and July, 1974. A plot of the monthly averages from those sites can be seen in Figures 3 and 4.

B. Soil

Unlike the air pollutant situation, there are several sources of soil pollution of significant magnitude. They include deposition of air and water-born contaminants and the physical incorporation of solid dumped contaminants. A listing of possible sources follows:

Air Born Sources

- dust blowing from other areas.
- deposition of particulate matter.

Water Born Sources

- deosition from polluted waters.
- deposition of flood residue.
- transport by surface runoff.

Contamination By Solids

- mine tailings disposal.
- jig tailings disposal.
- low grade ore disposal.
- ore and concentrate spills.
- tracking of material by transportation vehicles.

Because of this variety of sources, the level of observed lead concentrations in the soils of the valley is somewhat erratic. There are several small isolated areas throughout the valley that are observed as having high levels. Usually those high levels can be attributed to one or more of the above. Areas around old and present mills have high concentrations. Tailings disposal sites are high in lead and other metals. Flood plain sites show increased lead levels. Several other examples are common.

However, it is obvious that high levels are observed in the Kellogg-Smelterville area. Again, these concentrations are centered at the Bunker Hill complex. This is not surprizing as this area is the focal point of the lead industry in the valley. Certainly some of this soil contamination is a result of discharges and effluents from the smelter complex. Others are a result of ores and concentrates moving toward the smelter. Disposal of tailings and the use of mine wastes for fill, etc., contributes to this concentration. Essentially this area is, and has been, for over 80 years, the center of a large, active, and everchanging industry. The lead contamination in the soil of this area represents these many years of activity.

Figure 5 shows a plot of lead concentration in the soil versus the distance from the reference point.

C. Vegetation

The lead levels observed in vegetation are a product of both physiological uptake from contaminated soils and the absorption and deposition of particulate matter from the atmosphere. We would, therefore, expect that the vegetation would reflect the trends observed in the soil and air. Figure 6 reflects the validity of this suspicion. Vegetation lead levels also show a distinct relationship with distance from the reference point. Plotted on this graph are both washed and unwashed vegetation samples. Because these are all samples of grasses, there is no clear relationship between washed and unwashed vegetation except in areas where the particulate matter is heavy with lead.

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D. Summary

Generally, it is apparent that the center of abnormal lead concentrations in the valley is the Bunker Hill complex. Other small "hot spots" exist, notably around old mining and milling areas and the flood plains of creeks that service the same. Leaching and wind blown dusts from these areas still constitute an active source. Concentrate is still spilled by trains moving up and down the valley. However, in both magnitude and source strength, the largest and certainly what is expected to be the most significant active source of contamination is air-born emissions from the Bunker Hill complex. Nevertheless, it must be recognized that the relative public health significance of current emissions from the Bunker Hill complex and the lead deposited in the environment during past years has not yet been established.

III. PHASE II STATUS REPORT:

Since Phase II of the survey is not as directly related to specific lead absorption cases as is Phase III, laboratory analysis of samples taken in Phase II were assigned a lower priority than those taken in Phase III. Further, because of the nature of the Phase II study, data must be examined through the use of a computer. Therefore, a report of the Phase II information must be delayed.

IV. PHASE III STATUS REPORT:

The Phase III study involves concentrated environmental sampling to verify Phase I results and also is concerned with localized environmental sampling to correlate with blood samples taken from children. Since large numbers of samples were taken and laboratory capacity is limited, sampling areas were prioritized for analysis. At this point in time, only data for Area I (Smelterville area) are available. A detailed examination of the data has not yet been accomplished but average values and range of values for various sample types from Area I have been calculated and are shown below:

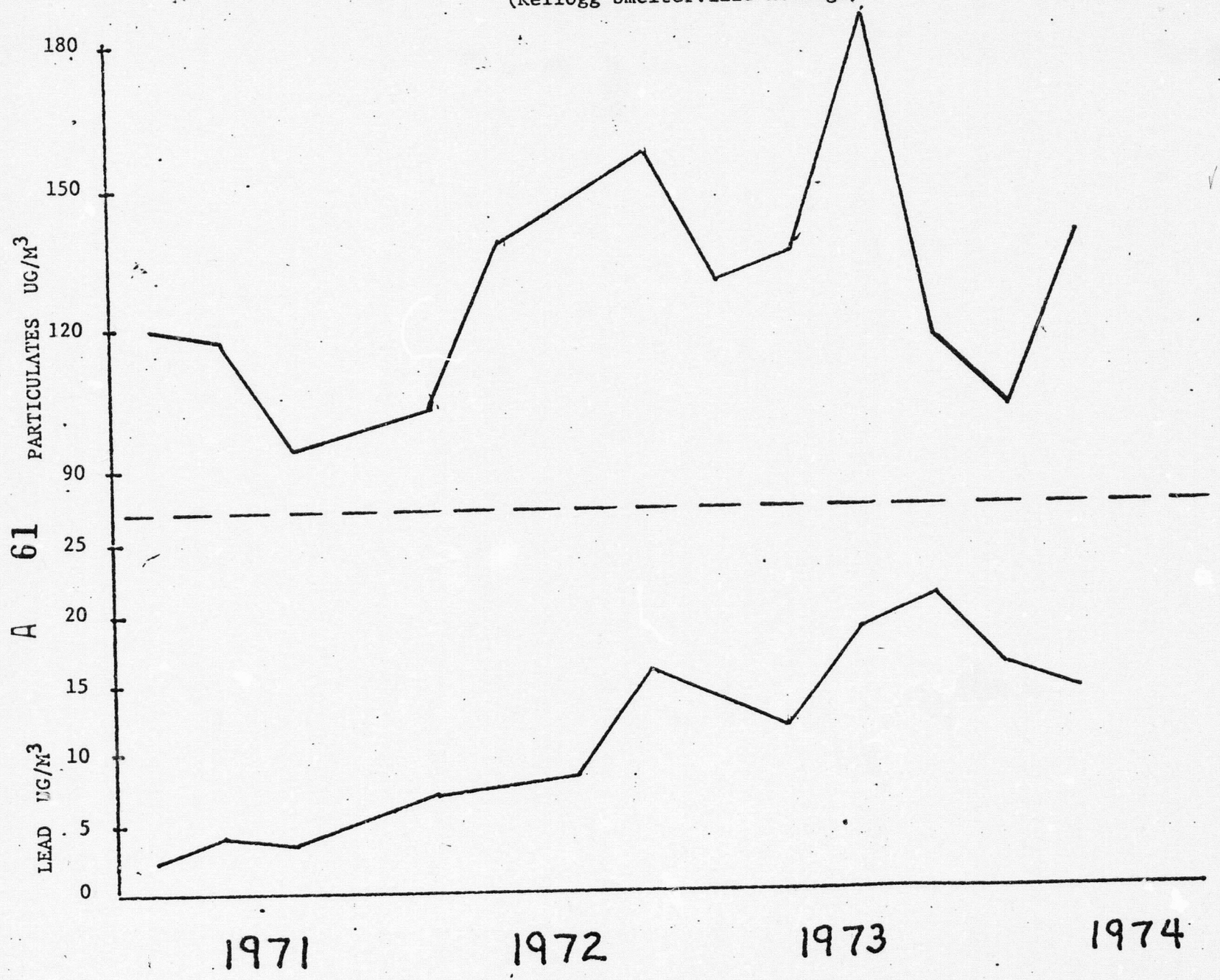
	LEAD CONCENTRATION	
	AVERAGE	RANGE
Soil	6,700 ppm	1,089 - 26,700 ppm
Vegetation (grass)	1,200 ppm	109 - 2,200 ppm
House dust	12,000 ppm	900 - 38,900 ppm
Inside paint	-	48 - 179,000 ppm
Outside paint	-	1,047 - 353,000 ppm

The lead concentrations in all of the sample types are sufficiently high to warrant concern. However, a cursory examination of the data shows no obvious correlation between levels of environmental lead and levels of blood lead in children. This is to be expected, however, since nearly all of the blood lead levels reported for Area I are high and the range of values is not great. Therefore, the possibility of correlation of blood and environmental lead data in Area I is limited. More comprehensive examination of Area I data may show subtle correlations. Later analysis of all samples from all geographic areas (which include a greater range of blood lead levels) may show obvious correlation and develop a cause-effect relationship.

FIG 1

AVERAGE QUARTERLY LEAD & PARTICULATE DATA

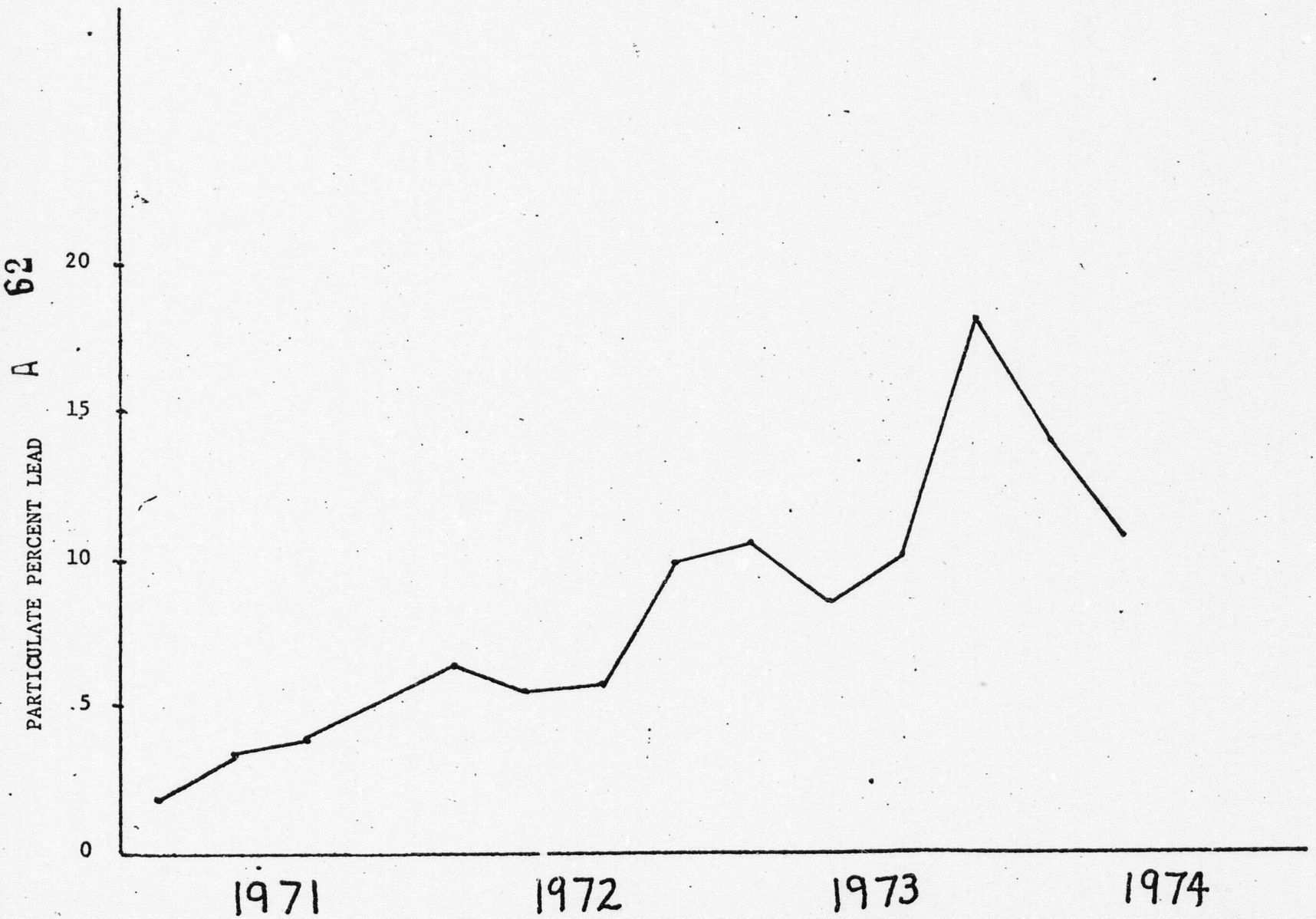
(Kellogg-Smelterville Average)



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FIG. 2

PERCENT Pb IN PARTICULATE (Kellogg-Smelterville Average)
(by Quarter)



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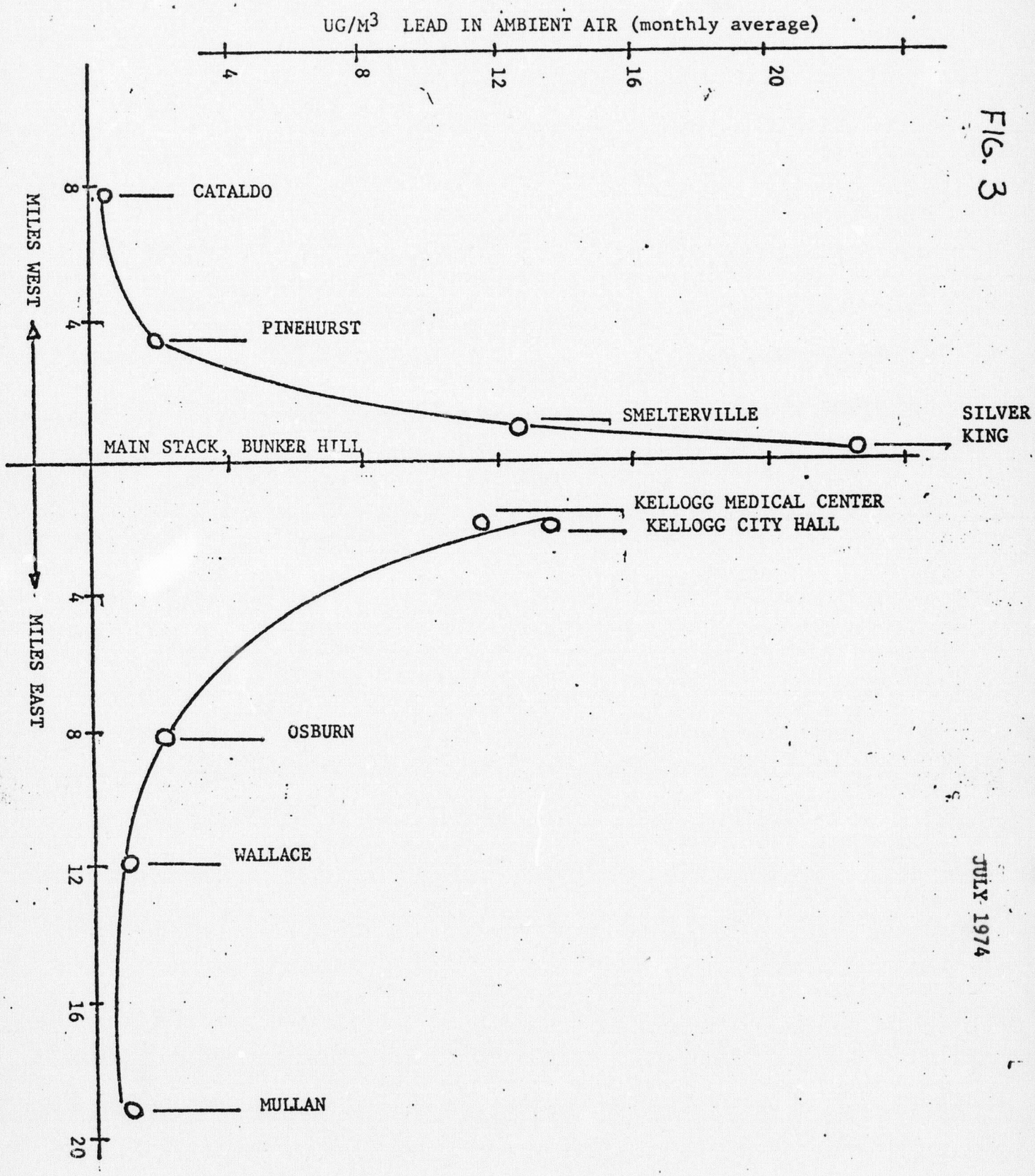


FIG. 3

JULY 1974

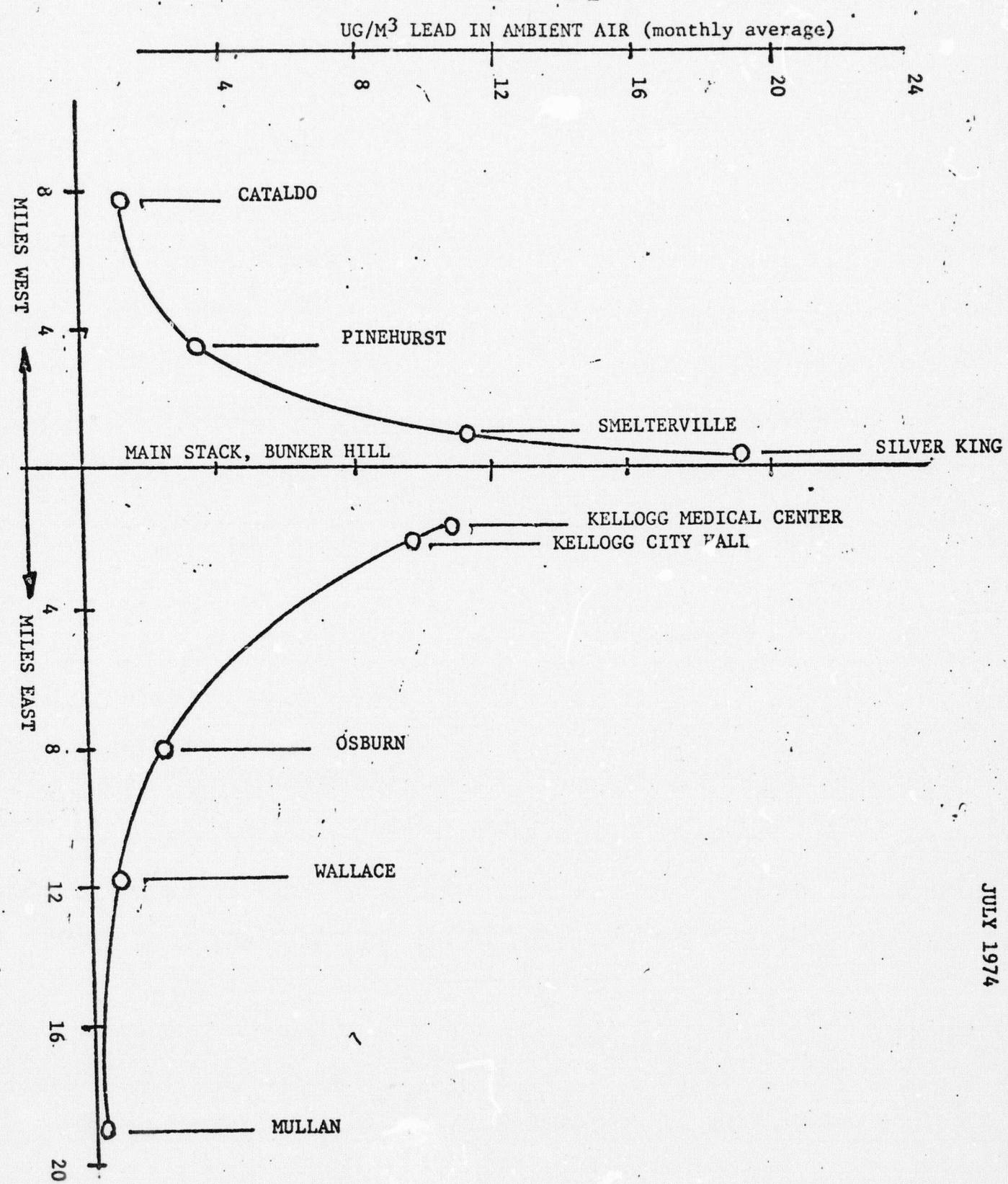


FIG 4

JULY 1974

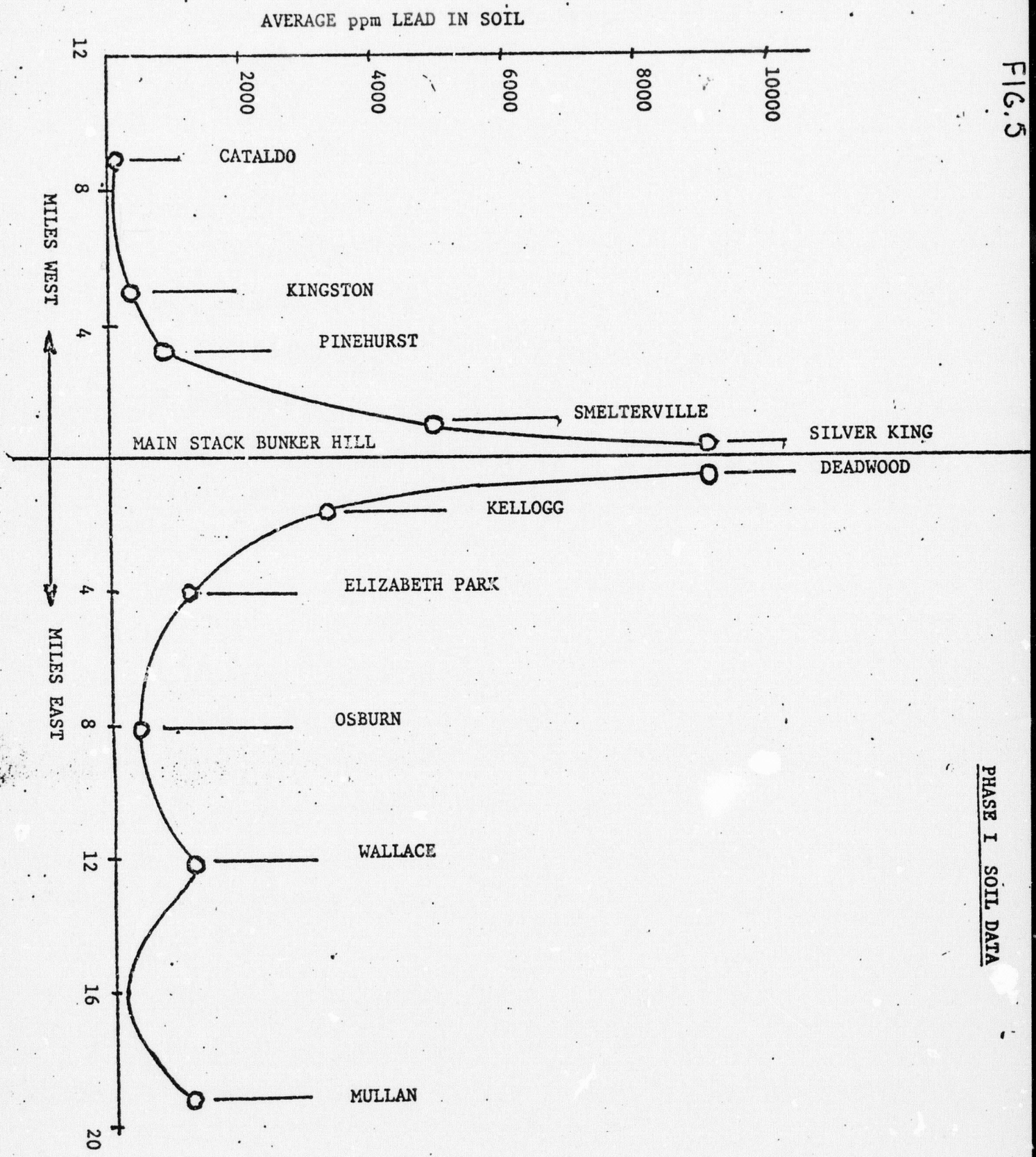
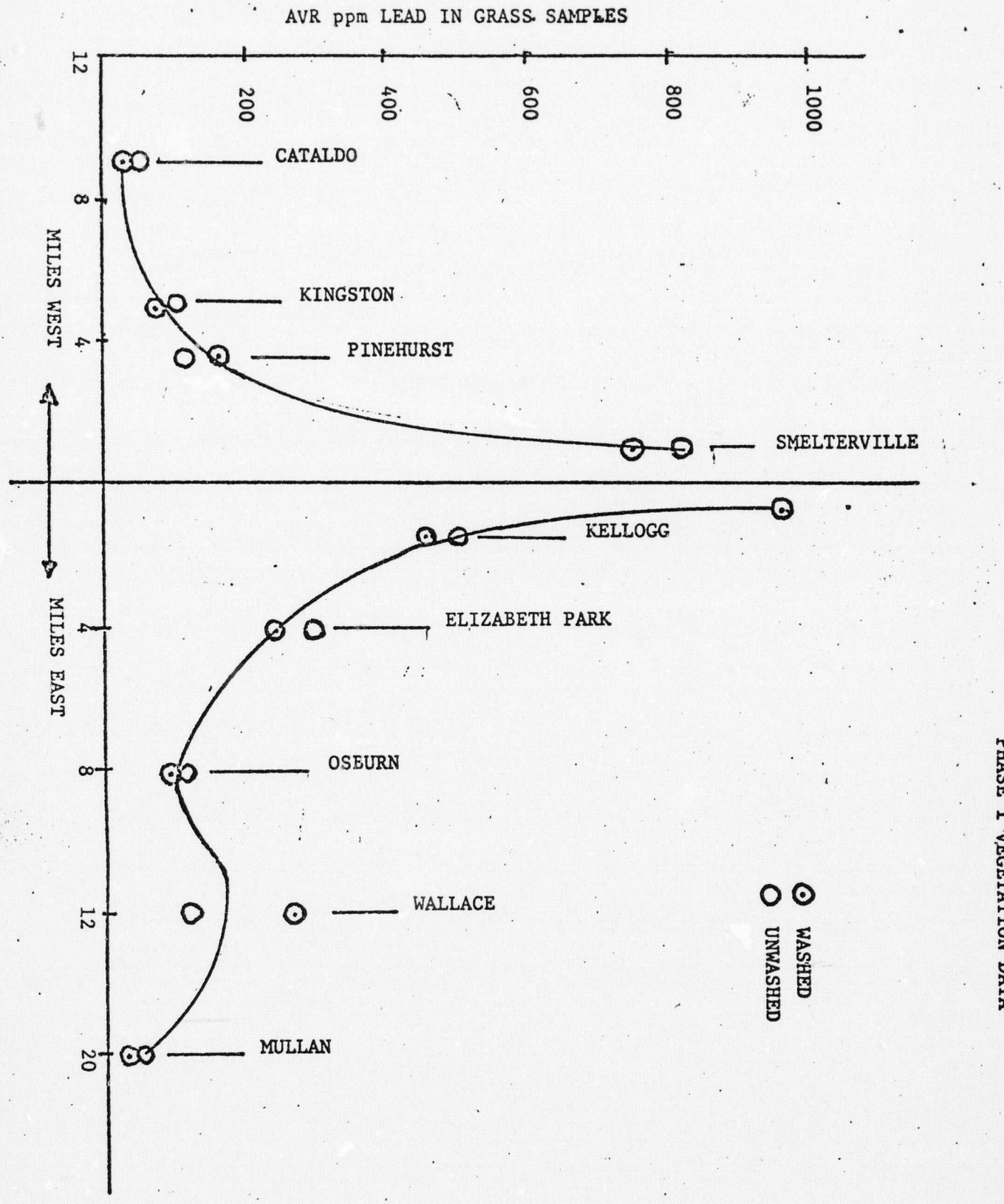


FIG. 5

PHASE I SOIL DATA



PHASE I VEGETATION DATA

FIG. 6

SILVER VALLEY LEAD STUDY - PHASE I SOIL SUMMARY

	tenths of miles distance from smelter	0° = N. Azimuth from smelter	Soil pH	(ppm) Soil Pb	PRIMARY Veg. Pb (ppm) Washed	Unwashed	MISC. VEG. Pb Washed	Unwashed
ty Park	6.6	276	4.5	5000	1075	1051	976	1051
ool	2.7	237	6.0	9000	-	-		
hool	118	114	6.9	1300	98	126	143, 23	119, 87
School	99.6	110	6.0	185	53	46		
	185	106	6.6	2800	18	39	17.9	38.6
k	185	105	5.9	500	30	38		
	33	267	5.5	530	91	71		
ck	14.8	107	4.2	5000	705	889	916	502
rd	23	112	4.0	1200	699	783	652	1010
e	15.6	89	4.4	3500	229	244		
n	10.5	86	4.2	1100	-	-		
	16.7	105	6.6	3000	388	561		
nool	16.7	75	5.9	800	271	272		
Park	49.8	112	7.6	250	55	128		
	118	111	6.2	1200	430	68		
d	185	106	5.9	1200	14	17		
	185	106	5.6	650	12	16		
	37.3	106	5.8	620	50	56		
h	31.1	96	-	1300	164	251		
	40.1	106	4.2	900	267	336		
yard	56.4	270	5.6	125	142	161		
	64.6	323	6.4	165	4.01	6.		
n	79.0	108	-	250	244	228		
	16.3	253	4.3	630	303			
	16.3	253	4.6	583	41	246	146	4896
	31.9	275	5.5	270	70	261		
	25.7	265	5.3	360	237	349		
	44.4	289	6.4	180	114	29		
	92.3	273	5.6	175	14	4.7		
	75.9	273	-	193	148	40		
	62.3	271	5.6	75	740	29		
	46.7	270	6.0	290	19	42		
Park	36.2	267	5.1	350	150	129		
11 Park	7.8	282	4.3	4000	-	-		
. End	12.5	271	4.0	5000	406	563		
	7.0	136	5.2	9000	950	3478	619	1339

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SILVER VALLEY LEAD STUDY - PHASE I SOIL SUMMARY

	tenths of miles distance from smelter	0° = N. Azimuth from smelter	Soil pH	(ppm) Soil Pb	PRIMARY Veg. Pb (ppm) Washed	Unwashed	MISC. VEG. Pb Washed	Unwashed
	20.2	99	4.6	2200	-	-		
	21.4	116	5.2	7000	-	-		
	19.5	140	6.0	6300	270	207		
	46.7	96	4.4	1000	94	111	148	185
	57.6	88	5.3	190	7	13		
	72.0	108	5.7	1300	56	90	109	142
gs	78.7	107	6.2	350	-	-		
l	78.7	107	5.5	130	23	85	64	163
k	99.7	110	6.5	300	32	63		
on	79.0	107	4.9	950	212	278		
	40.9	249	5.2	1340	<11	<11	35, 45	55, 86
	44.4	267	5.0	200	32	45		
	43.6	265	5.5	200	14	18		
	47.5	265	4.7	205	23	47		

SUPPLEMENTARY HEAVY METAL SCREEN - PHASE I (ppm)

Sample #	Type	Cadmium Cd	Arsenic As	Mercury Hg	Zinc Zn	Antimony Sb	
2	Soil	61	72.1	48.1	5953	-	
3	Soil	9	10	<1.5	110.5	-	
5	Soil	8	9	<1.5	1840	-	
10	Soil	-	-	-	-	82	
14	Soil	-	-	-	-	366	
17	Soil	-	-	-	-	66	
19	Soil	-	-	-	-	78	
21	Veg	3.5	< 1.2	<1.5	83	-	(washed)
21	Veg	4.0	< 1.2	<1.5	89	-	(unwashed)
21	Soil	2.6	10	<1.5	110	-	
22	Soil	-	-	-	-	52	
25	Soil	-	-	-	-	66	
25	Veg	<2	<1.2	<1.5	61	-	(washed)
25	Veg	3	<1.2	<1.5	98	-	(unwashed)
26	Veg	46	<1.2	<1.5	123	-	(unwashed)
26	Veg	<2	<1.2	<1.5	100	-	(washed)
26	Soil	4.5	8.0	<1.5	155	-	
28	Soil	-	-	-	-	52	
29	Soil	2.5	8.6	<1.5	160	-	
33	Soil	3.9	9.0	<1.5	219	-	
37	Soil	10.1	34.6	<1.5	1100		
40	Soil	-	-	-	-	72	
43	Soil	-	-	-	-	60	
44	Soil	4.2	11.8	<1.5	160		
45	Soil	-	-	-	-	64	
47	Soil	-	-	-	-	276	
50	Soil	-	-	-	-	60	

A 70

PHASE I PART C DATA

Garden Survey - (Silver Valley)

(Area Average - PPM (Dry Basis)

	CATALDO		KINGSTON		PINEHURST		SMELTERVILLE		SILVER KING		KELLOGG		OSBURN		WALLACE	
	Pb	Cd	Pb	Cd	Pb	Cd	Pb	Cd	Pb	Cd	Pb	Cd	Pb	Cd	Pb	Cd
ETTUCE																
Washed	6	4	16	6	130	9	106	15	611	60	91	15	24	6	28	11
Unwashed	17	5	41	6	188	9	770	35	2 270	58	293	17	305	18	47	8
EETS																
Washed	6	2	7	2	13	7	170	28	513	41	104	19	13	3	16	5
Unwashed	9	2	11	2	94	9	277	31	617	44	232	16	23	6	150	10
ARROT																
Washed	6	2	6	2	10	5	160	7	26	16	20	7	6	5	6	3
OIL	177	3	141	4	950	9	3 040	44	4 100	47	1 251	10	1 775	16	561	14

UNITED STATES DISTRICT COURT
FOR THE SOUTHERN DISTRICT OF NEW YORK
----------x

NATURAL RESOURCES DEFENSE COUNCIL, INC.
15 West 44th Street
New York, New York 10036

NEW YORK ASSOCIATION FOR BRAIN INJURED CHILDREN
95 Madison Avenue
New York, New York

CENTER FOR SCIENCE IN THE PUBLIC INTEREST
1779 Church Street, NW
Washington, D.C.

ENVIRONMENTAL ACTION COALITION
1346 Connecticut Avenue, NW
Washington, D.C.

FRIENDS OF THE EARTH
620 C Street, SE
Washington, D.C.

AFFIDAVIT

HIGHWAY ACTION COALITION
Room 731, DuPont Circle Building
Washington, D.C.

NATIONAL WELFARE RIGHTS ORGANIZATION
1420 N Street, NW
Washington, D.C.

Plaintiffs,

v.

RUSSELL TRAIN, As Administrator
Environmental Protection Agency
4th & M Streets, SW
Washington, D.C.

U.S. ENVIRONMENTAL PROTECTION AGENCY
4th & M Streets, SW
Washington, D.C.

Defendants.

----------x

STATE OF NEW YORK)
: SS
COUNTY OF NEW YORK)

WILLIAM SHAPIRO, being duly sworn, deposes and says:

1. I am an assistant to the Commissioner of the New York City Department of Air Resources. I am responsible for various technical programs for the Bureau of Motor Vehicle

Pollution Control including the implementation of New York City's lead reduction law (section 1403.2-13 of the Administrative Code of the City of New York). I hold a Bachelor's Degree cum laude in Chemical Engineering from City College of New York, and a Master's Degree in Chemical Engineering with a minor in Air Pollution from New York University. This affidavit is submitted in support of plaintiff's motion for summary judgment in this proceeding.

2. New York City's lead regulations provided for a gradual reduction and eventual elimination by January 1, 1974 of lead additives in gasoline sold in the City.

		95.9 Octane No. and above	Below 95.9 Octane No.
I	On & after Nov.1, 1971	2.0 grams per gal	1.5 grams per gal
II	On & after Jan.1, 1972	1.0 " " "	1.0 " " "
III	On & after Jan.1, 1973	o.5 " " "	0.5 " " "
IV	On & after Jan.1, 1974	zero (0.075) grams	zero (0.075) grams

This schedule has been modified, however, in the course of litigation between the City and five oil companies. A primary issue in this litigation involves the pre-emption of these local lead regulations by less stringent federal lead regulations. With respect to these five oil compamies, which supply approximately 51% of New York City's gasoline supply, the 1972 limit, 1.0 grams/gal, has to date remianed in effect.

3. The following reductions in the concentration of lead in the ambient air have been measured by personnel of the Department of Air Resources at thirty-seven locations in New York City.

(Micrograms/Cubic Meter)

	Average	% Reduction From 1970	Average of Peaks	% Reduction from 1970
1970	1.93		9.64	
1971	1.49	-22.8%	9.70	
1972	1.28	-33.7%	5.43	-43.7%
1973 (1st Nine Months)	1.03	-46.6%	2.94	-69.5%

New York City's lead regulations, in spite of their modification, have improved the daily average air quality with repsect to lead by 46%, and the average of daily peak concentration by 69%.

4. Pre-emption of New York City's lead additive regulations by federal regulations (38 Fed. Reg. No. 234, page 33734 et seq.) will cause a serious deterioration of the air quality in New York City with respect to lead. Under the federal regulation effective for 1975 (1.7 grams/gallon for all grades including unleaded gasoline) ambient levels of lead concentration will necessarily rise in New York City back to approximately the 1971 levels. Not until 1978 will the federal regulations again achieve the reduced level of lead in the ambient air that New York City first attained in 1973. In 1979, the final stage in the federal regulations, .5 grams per gallon is still permitted, compared to zero grams per gallon in New York City's lead regulations. Thus, to avoid dangerous peak concentrations in the area of high traffic density the elimina- of lead additives in gasoline is necessary. Protection from pre-emption of New York City's more stringent regulations is therefore important to avoid degradation and to permit further improvement of the City's air quality.

William Shapiro
WILLIAM SHAPIRO

Sworn to before me this
12th day of November, 1974
Melvin L. Ortner
Notary Public

MELVIN L. ORTNER
Notary Public, State of New York
No. 24-4517643
Certificate Filed in Kings County
Term Expires March 30, 1976

UNITED STATES DISTRICT COURT
FOR THE SOUTHERN DISTRICT OF NEW YORK

----------X

NATURAL RESOURCES DEFENSE COUNCIL, INC., et al.,

Plaintiffs,

v.

RUSSELL TRAIN, Administrator, U.S. Environmental Protection Agency, et al.,

Defendants.

CIVIL ACTION NO. 74-4617

AFFIDAVIT

----------X

STATE OF NEW YORK)
: ss.:
COUNTY OF NEW YORK)

MARCIA ELIZABETH TOMPKINS, being duly sworn, deposes and says:

1. I am employed by the Natural Resources Defense Council, Inc., as a legal secretary; I am over 21 years of age; and, I live at 77 East 12th Street in New York City.

2. On Thursday, November 14, 1974, I attended the afternoon session of the bi-monthly meeting of the U.S. Environmental Protection Agency ["EPA"] National Air Quality Criteria Advisory Committee ["the Committee"] meeting held in Arlington, Virginia.

3. Ms. Donna Wicker of the office of the Chairman of the Committee advised me that minutes of the meeting are not now available, and, I am basing this affidavit upon notes taken by me throughout the meeting and my recollections.

4. Originally established by the Department of Health, Education and Welfare in 1968, the Committee was reconstituted by Order No. 1130.8 of the EPA Administrator [attached hereto as Exhibit A]. That Order states, inter alia:

"The Committee was established in order to obtain assistance [to the EPA Administrator] in the development and implementation of the purposes of the Clean Air Act, as amended, regarding air quality criteria and specifically with respect to:

a. The development and issuance of air quality criteria under section 108(a)(2);

...

c. The revision, from time to time, of the list of air pollutants which meet the requirements for issuance of air quality criteria under section 108(a)(1); ...." (Order, ¶3, p. 1)

5. The Committee is composed of emminent scientists and laypersons who are affiliated with universities, industry, and government. [The Committee membership is attached hereto as Exhibit B.]

6. One of the items on the Committee's November 14, 1974, agenda was the issue of whether ambient lead should be listed as a criteria pollutant under section 108(a)(1) of the Clean Air Act. [The Agenda is attached as Exhibit C.] The Committee had taken up the issue of listing lead, on its own initiative, as a result of concern raised at previous meetings as to the adequacy of EPA's approach to safeguarding health from lead pollution.

7. In order to help it reach a resolution on whether to recommend the listing of ambient lead, the Committee invited members of the Agency and public interest groups to make presentations to the Committee at the meeting. [The Memo to the Committee from B.J. Steigerwald, Director of EPA's Office of Air Quality Planning and Standards, presented to the Committee by Mr. Don Goodwin, Director of EPA's Emission Standards and Engineering Division, is attached hereto as Exhibit D; the presentation to the Committee by the Natural Resources Defense Council, Inc., is attached as Exhibit E.]

8. After considerable discussion of the Steigerwald Memo, presented by EPA's Mr. Goodwin, Dr. Samuel Epstein, a member of the Committee, remarked that the Agency appeared to be "seriously misleading" the Committee.

9. The Committee then questioned representatives of public interest groups, including Mr. David Hawkins, an attorney employed by the Natural Resources Defense Council.

10. A discussion then followed by the Committee on the merits and the feasibility of setting an ambient standard for lead. Members of the Committee noted that the Committee had, many times, discussed and was very familiar with the various data on the health hazards of ambient lead and the nature of EPA's approach to the problem of lead pollution; that it had also many times discussed the feasibility of listing ambient lead for the purpose of establishing national primary and secondary air quality standards; and that a final resolution of that subject matter was in order. Concluding the discussion, Dr. Haagen-Smit, Chairman of the Committee, noted his especial concern for young children, both in urban areas and living around isolated smelters, who needed more stringent health protection from the health hazards of ambient lead than that presently afforded by EPA.

11. Members of the Committee then proposed the following resolution for a Committee vote:

> "MOVE that the Committee formally request that the Administrator of the United States Environmental Protection Agency list ambient lead as a pollutant under section 108 of the Clean Air Act for the purpose of establishing ambient air quality standards for lead."

12. After noting the abstention from voting of one member of the Committee, a vote was called and the above resolution was adopted unanimously by all members of the Committee, after which the meeting was adjourned.

Marcia Elizabeth Tompkins

Sworn to before me this
21st day of November, 1974

Angus Macbeth

ANGUS MACBETH
Notary Public, State of New York
No. 31-7648280
Qualified in New York County
Commission Expires March 30, 1975

ENVIRONMENTAL PROTECTION AGENCY

A 78

ORDER

1130.8

January 5, 1973

ORGANIZATION AND FUNCTIONS - COMMITTEES, BOARDS, PANELS, AND COUNCILS

NATIONAL AIR QUALITY CRITERIA ADVISORY COMMITTEE

1. PURPOSE. This Order revises the National Air Quality Criteria Advisory Committee charter to include requirements of section 9(c) of P.L. 92-463, Federal Advisory Committee Act, dated October 6, 1972. The Office of Research and Monitoring provides the necessary support for the Committee.

2. AUTHORITY. The Committee was established by the Surgeon General, U.S. Public Health Service, Department of Health, Education, and Welfare, on March 4, 1968, under section 110(d) of the Clean Air Act, as amended, and reestablished by the Administrator, Consumer Protection and Environmental Health Service, DHEW, pursuant to the Secretary's Reorganization Order dated July 1, 1968; transferred to the U.S. Environmental Protection Agency pursuant to Reorganization Plan No. 3, December 2, 1970; reconstituted by the Administrator of EPA on October 5, 1972, pursuant to section 117(d) of the Clean Air Act, as amended.

3. OBJECTIVES AND SCOPE OF ACTIVITY. The Committee was established in order to obtain assistance in the development and implementation of the purposes of the Clean Air Act, as amended, regarding air quality criteria and specifically with respect to:

a. The development and issuance of air quality criteria under section 108(a)(2);

b. The review and, as appropriate, modification and reissuance of air quality criteria under section 108(c);

c. The revision, from time to time, of the list of air pollutants which meet the requirements for issuance of air quality criteria under section 108(a)(1);

d. The listing of a substance as a hazardous air pollutant under section 112(b)(1)(A); and

e. The determination of adverse effects on the public health and welfare of air pollutants controlled or under consideration for control under applicable provisions of the Clean Air Act, as amended, including the determination, under section 211(c)(1)(A), that the emission product of a fuel or fuel additive will endanger the public health or welfare.

The Committee will continue to function as long as there is a need to carry out these objectives. The estimated annual operating cost of the Committee totals $52,000, which includes 1.3 man-years staffing requirement.

4. FUNCTIONS. The Committee provides advice and consultation to the Administrator, through the Assistant Administrator for Research and Monitoring, regarding (a) the development, documentation, and revision of air quality criteria for specific pollutants; (b) the designation of a substance as a hazardous air pollutant; and (c) the effects on the public health and welfare of other pollutants, not covered by air quality criteria or listed as hazardous, and of airborne emission products of motor vehicle fuels and fuel additives.

5. COMPOSITION AND MEETINGS. The Committee consists of the Assistant Administrator for Research and Monitoring, or his designee, as Chairman, and 15 members appointed by the Administrator, for overlapping terms of three years. Members are prominent scientists and/or administrators concerned with the quality of the environment and resident in universities, State and local governments, research institutions, or industry. They shall be selected from relevant disciplines and for their expertise in the establishment of air quality criteria and/or in the interpretation of scientific evidence indicative of adverse effects of atmospheric pollutants. Meetings are held six times a year, as called by the Chairman. A full-time salaried officer or employee of the Agency will be designated as Executive Secretary who will be present at all meetings and is authorized to adjourn any such meeting whenever he determines it to be in the public interest.

6. DURATION. The Committee will terminate two years from the filing date of this Charter unless extension beyond that date is authorized in accordance with section 14 of P.L. 92-463.

William D. Ruckelshaus

William D. Ruckelshaus
Administrator

A 80

National Air Quality Criteria Advisory Committee

November 14, 1974

Participants
(Anticipated)

Chairman

Dr. Arie J. Haagen-Smit
Director
Plant Environment Laboratory
California Institute of Technology

Members

Dr. Mary O. Amdur
Harvard University

Dr. David M. Anderson
Bethlehem Steel Corporation

Dr. Anna M. Baetjer
The Johns Hopkins University

Dr. Thomas D. Crocker
University of California

Dr. Samuel S. Epstein
Case Western Reserve University

Dr. Arie J. Haagen-Smit
California Institute of Technology

Dr. Eugene P. Odum
University of Georgia

Mr. Elmer Robinson
Washington State University

Mr. Morton Sterling
Wayne County, Michigan

Mr. Arthur C. Stern
University of North Carolina

Mr. Elmer P. Wheeler
Monsanto Company

Dr. John T. Wilson, Jr.
University of Washington

Executive Secretary

Mr. Ernst Linde
National Environmental Research Center
Research Triangle Park, North Carolina

EXHIBIT C

NATIONAL AIR QUALITY CRITERIA ADVISORY COMMITTEE

A 81

Conference Room A
Crystal Mall Building No. 2
Arlington, Virginia

November 14, 1974

A G E N D A

9:00 a.m.	1.	Opening Remarks	Dr. Haagen-Smit
	2.	Items of Interest from EPA	Dr. Hueter
	3.	Vinyl Chloride	
		- Presentation of Briefing Report on "Environmental Aspects of Vinyl/Polyvinyl Chloride" (External Draft dated October 15, 1974)	Dr. Bridbord
		- Status of Regulatory Effort	Mr. Goodwin
		- Discussion	
		- Recommendations	
12:15 p.m.		L U N C H	
1:30 p.m.	4.	Airborne Lead	
		Is There a Need for National Ambient Air Quality Criteria (and Standards)?	
		- Introductory Remarks	Dr. Haagen-Smit
		- Control Strategy Considerations	Mr. Goodwin
		- Discussion	
		- Recommendations	
3:30 p.m.	5.	Other Business, Concluding Remarks and Date and Place of Next Meeting	

A D J O U R N M E N T

EXHIBIT D

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY

A 82

SUBJECT: Preferred Strategy for Regulating Atmospheric Lead Emissions From Stationary Sources

DATE: NOV 07 1974

FROM: B. J. Steigerwald, Director
Office of Air Quality Planning and Standards

B. J. Steigerwald

TO: Ernst Linde, Executive Secretary
National Air Quality Criteria Advisory Committee

From emission inventory estimates, it is clear that the major lead emission sources, in terms of either mass or population affected, are mobile. Only 10 percent of total nationwide lead emissions evolve from stationary sources, and any stationary source control strategy must recognize the much greater importance of mobile source control.

The basic mobile source control strategy, both for new and existing vehicles, is one of national performance standards. As the preamble to regulations implementing a phased reduction in the lead additive content of leaded gasoline states:

> "...the Administrator determined that it (is) difficult, if not impossible, to establish a precise level of airborne lead as an acceptable basis for a control strategy..."

The reasons that it is difficult, if not impossible, to establish an ambient air quality standard to regulate lead emissions from mobile sources are the multiple sources of lead exposure (paint, food, airborne lead, etc.) and the multiple routes of exposure to airborne lead (inhalation and ingestion); this same reasoning applies to stationary sources.

The Office of Air Quality Planning and Standards (OAQPS) began its preferred standards path analysis for the control of stationary sources of lead before the Administrator reached his conclusions concerning mobile sources. The Office of Research and Development did prepare materials concerning public health and welfare effects, which have been distributed to the Committee members. If the Administrator had chosen to define a safe ambient level as a basis for lead additive control, OAQPS might have decided in favor of National Ambient Air Quality Standards (NAAQS). At issue, however, would be the disruptive effects of NAAQS on EPA's national fuel additives program. This program, promulgated December 6, 1973, requires a phased lead additive reduction that will decrease emissions to one quarter of the current level by 1979 and preempts local fuel additive regulations. If a NAAQS for lead were promulgated, it would supersede the preemption and a diversity of fuel requirements which petroleum refineries would find difficult to supply could be established. The required production and distribution of a variety of lead-content

EXHIBIT E

gasolines would be highly undesirable. Also, the fuel additive program is based on a five-year phased reduction; achievement of NAAQS is mandated as a three-year program in the Clean Air Act. This difference in timing could create difficult regulatory problems.

A second option considered but rejected by OAQPS for regulating lead is National Emission Standards for Hazardous Air Pollutants (NESHAP). The health effects data contained in the draft document Summary Report on Lead From Stationary Sources which was reviewed by the National Air Quality Criteria Advisory Committee (NAQCAC) indicate that lead emissions should be regulated, but they do not pose a threat to human health to the extent of causing or contributing "to an increase in mortality or an increase in serious irreversible, or incapacitating reversible, illness." Elevated blood lead levels have been observed in the vicinity of lead-emitting sources, but these levels have not exceeded 40 ug lead/100 g of blood except in two locations. [A blood lead level of 40 ug lead/100 g blood has been established in EPA's Position on the Health Implications of Airborne Lead as a guideline to indicate a level of lead intake which should be prevented. Blood lead levels in the range of 40 to 60 ug/100 g have been associated with undesirable subclinical biochemical changes.] The blood lead levels elevated above 40 ug/100 g were observed in the vicinity of two primary lead smelters and are attributable largely to unusual topographical and meteorological conditions. Individual action is being taken at both of these locations to reduce exposure levels. The lead additive regulations support the conclusion that NESHAP is not the appropriate regulatory route for lead; they require a phased reduction in the lead content of gasoline over several years rather than immediate reduction to some "safe" level or outright elimination. Furthermore, Section 112 of the Clean Air Act (NESHAP) contains no authority for establishing emission standards for protection of welfare. Animals are susceptible to lead poisoning at lower emission rates than humans because animals consume unwashed lead-containing vegetation. Emission standards for the protection of health would not necessarily protect welfare.

The regulatory route selected by OAQPS, Standards of Performance for New Sources (SPNS), can be justified since lead emissions do contribute to the endangerment of health or welfare, as evidenced by the elevated blood lead levels observed in the vicinity of stationary sources. SPNS are similar to the mobile source control strategy, and provide an effective means for minimizing lead emissions to the environment from new and existing facilities by requiring best control technology (considering cost). SPNS for particulate matter (a criteria pollutant) rather than for lead (a designated pollutant) could be used to limit

particulate lead emissions from new sources only. However, SPNS for lead will have a larger impact on emissions since States will be required to regulate existing sources as well.

A study of the impact SPNS for lead emissions would have concluded that standards for two lead-emitting industries are definitely merited (lead-acid storage battery manufacture and gasoline additive manufacture), that one industry deserves further study (melting of type metal), that future practices in a fourth source should be carefully monitored (combustion of waste crankcase oil), and that a substantial effort should be made to quantify and control fugitive lead emissions.

Therefore, a limited but specific program of SPNS for selected industries is recommended as the preferred regulatory route for controlling stationary sources of lead emissions. A more detailed discussion of this program and the reasons for regulating lead with SPNS rather than NAAQS or NESHAP is contained in the draft document entitled Preferred Standards Path Analysis on Lead Emissions from Stationary Sources which was distributed to the NAQCAC for review.

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NATIONAL AIR QUALITY CRITERIA ADVISORY COMMITTEE

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY

Committee Meeting

November 14, 1974

STATEMENT AND QUESTIONS

By the Natural Resources Defense Council, Inc.

RE: NATIONAL PRIMARY AND SECONDARY AIR QUALITY STANDARDS FOR AMBIENT LEAD

David Hawkins
Marcia Tompkins
(Natural Resources Defense Council, Inc.)
1710 N Street, NW
Washington, D.C. 20036

15 West 44th Street
New York, New York 10036

November 14, 1974

STATEMENT

A. THE HEALTH EFFECTS OF AIRBORNE LEAD.

EPA acknowledges the toxicity of airborne lead and has found that:

a. Lead poisoning results in death or severe illness, including irreversible brain damage. EPA's Position on the Health Implications of Airborne Lead at III-1 to 5 (Nov. 28, 1973).

b. Lead exposure insufficient to cause overt symptoms alters vital bodily functions, such as the production of hemoglobin. Id. at III-9 to 10. On the basis of such effects, EPA has defined as unacceptable blood lead levels (id. at III-11), which are commonly exceeded. Id. at VII. While medical science has done little to investigate these alterations in bodily functions (id. at III-3), such subclinical exposure has been associated in children with subtle damage to the nervous system (including hyperactivity and loss of motor control), kidneys, and other systems. Id. at III-5 to 11.

c. Airborne lead has resulted in street dust in cities, such as Washington, D. C., and Boston, containing up to one percent lead (id. at II-6), equivalent to dangerous lead paint.

d. A quarter of urban children tested and millions of adults have unacceptable blood lead levels due to lead additives. Id. at VII-Tables. Humans absorb airborne lead from air breathed, water and food consumed, and dust and dirt pervading the metro-

politan environment. Id. at V, VII, VII-3. This absorbtion, on top of naturally occurring lead in food, causes unacceptable blood lead levels in the children, "a small, but significant" portion of all adults, and an important portion of the large broups of workers, such as garage mechanics and policemen, occupationally exposed to car fumes. Id. at VII and Tables.

e. Airborne lead is implicated in clinical lead poisoning. "For a child with pica for paint, the combination of the ingestion of a few chips of paint and an increased intake of lead from contaminated dusts would provide a total lead intake sufficient to cause systematic illness." Id. at VI-6. In addition, as many as half the cases of childhood lead poisoning cannot be traced to lead paint (id. at VI-12 to 13), so that lead dusts may be "an important contributing factor." Id. at VI-13. Since "young children mouth and actually eat things that are not food rather indiscriminately" ... "dustfall from airborne lead, if swallowed, can make a significant contribution to a small child's total lead intake and thereby contribute to the occurrence of lead poisoning, especially in urban areas." Id. at VI-20 to 21.

B. THE STRUCTURE OF THE CLEAN AIR ACT.

The Clean Air Act aims to "speed up, expand, and intensify the war against air pollution in the United States with a view to assuring that the air we breathe throughout the Nation is wholesome once again." H.R. REP. No. 91-1146, 91st Cong., 2d Sess. 1 (1970)

(emphasis added). To ensure this aim and to prevent the delays that had undercut previous clean air legislation, Congress incorporated health protection procedures that locked the Administrator into protecting people on a timetable. Sections 108-110.

The Act required that health be protected from ambient lead no later than May 29, 1976, since section 108(a)(1) required the initial list of pollutants be promulgated on January 30, 1971, 30 days after enactment.

The Summary and Follow-up Report of this Committee's June 27, 1974, meeting told of the "disagreement" by "some members" of the Committee with, inter alia, a recommendation which stated that "national ambient air quality standards for lead are 'not considered appropriate'". (Summary & Follow-up Report, p. 13, re: draft Summary Report on Lead from Stationary Sources). The Report summarized this disagreement as follows:

> "It was pointed out that airborne lead is ubiquitous, that its presence in the ambient air results from numerous and diverse sources and that it appeared, therefore, to meet the requirements for issuance, at the appropriate time, of air quality criteria for the purpose of establishing national primary and secondary air quality standards." (Summary and Follow-Up Report at 13.)

The Natural Resources Defense Council agrees with this statement and believes that ambient lead meets all the requirements for a pollutant to be listed both at the time of enactment and at all

times thereafter.

However, the Environmental Protection Agency failed to place ambient lead on the initial list pursuant to section 108(a)(1); has failed to list lead subsequently; and has taken no action to curb lead pollution under the health protection procedures of sections 108-110 of the Clean Air Act.

C. THIS FAILURE MAY NOW CAUSE SPECIAL HARM TO NEW YORK CITY.

While EPA's failure to establish national primary and secondary air quality standards for ambient lead threatens the public health and welfare generally, this failure will result in special harm to New York City by causing the tripling in the amount of lead contained in gasoline sold in the City starting January 1, 1975.

Under the New York City Administrative Code (§1403-2-13-11), the amount of lead contained in the gasoline sold in the City has been reduced by steps since 1971. When the federal fuel additive regulations become effective on January 1, 1975, section 211(c)(4) of the Clean Air Act requires that the City's ordinance be preempted.

Section 211(c)(4)(C) provides, however, that such preemption will not occur if a non-federal regulation is necessary to achieve a national ambient air standard. Thus, if ambient lead is listed and Title I procedures put in motion to commence setting ambient standards for lead, the City's ordinance will not be preempted,

since the federal regulations, which do not begin to reduce the lead content of gasoline until 1977, are insufficient to achieve any conceivable national air standard throughout New York City in 1975.

QUESTIONS

Therefore, the Natural Resources Defense Council believes it would be an important contribution towards resolving this controversy and fulfilling the mandate set forth in the Clean Air Act if this Committee would address three questions today:

1) Will this Committee today officially take the position and convey to the EPA Administrator its belief that ambient lead must be listed by the Administrator for the purpose of establishing national primary and secondary air quality standards?

2) If this Committee so takes an official position that ambient lead meets all the requirements to be listed by the Administrator, is not now the "appropriate time" to begin the Title I process of establishing national primary and secondary air quality standards for lead?

3) If this Committee to decides that (1) ambient lead must be listed and (2) that the appropriate time is now to begin the process of establishing national primary and secondary air quality standards for lead, would this Committee, therefore, officially ask the Administrator of the Environmental Protection Agency to:

a) list lead as a pollutant under section 108(a)(1) of the Clean Air Act? and

b) take such steps as are necessary to prevent preemption of New York City's local lead-in-gas regulations until such time as the Administrator issues national primary and secondary air quality standards for lead and determines to what extent the ambient air standard requires New York City's regulation of lead in gasoline?

UNITED STATES DISTRICT COURT

FOR THE SOUTHERN DISTRICT OF NEW YORK

- x

NATURAL RESOURCES DEFENSE COUNCIL, INC., et al.,

Plaintiffs,

v.

RUSSELL TRAIN, as Administrator of the U. S. Environmental Protection Agency, et al.,

Defendants.

CIVIL ACTION NO. 74-4617

- x

STATEMENT OF MATERIAL FACTS AS TO WHICH THERE ARE NO GERMANE ISSUES OF FACT TO BE TRIED

I. PARTIES

A. Plaintiffs

1 The Natural Resources Defense Council, Inc. (NRDC) is a non-profit, membership corporation organized under the laws of the State of New York. NRDC's principal office is at 15 West 44th Street, New York, New York 10036. It also has offices in Washington, D. C., and Palo Alto, California. NRDC has 12,000 members and contributors throughout the country, of whom 1,800 live in New York City. NRDC's purposes, which are supported by its members and contributors, are to enhance and protect environmental quality, to monitor federal, state, and city agencies responsible for implementing environmental legislation, and, when necessary, to litigate the adequacy of agency actions implementing such statutes.

2 The New York Association for Brain Injured Children (NYABIC) is a not-for-profit corporation formed under the laws of the State of New York. NYABIC and its local chapters have 3,200 member families, who are virtually all parents of brain injured children. The NYABIC chapters in New York City have a total

membership of 1,100 families. These members of the New York City chapters live within the City of New York.

Brain injury is a neurological disorder, which impairs the ability of children with normal intelligence to learn. Its symptoms are hyperactivity, inability to concentrate, poor impulse control, and difficulty learning to read and write. Lead poisoning, which children can contract by eating lead paint and lead-laden dust, and by breathing ambient lead is a major cause of brain injury.

NYABIC's purpose as an organization is to promote the interests of brain injured and learning disabled children, both by obtaining better services for those children who already suffer the consequences of brain damage and by preventing future brain damage to other children. In furthering its purposes, NYABIC has worked with government agencies to provide more educational and medical services to brain injured children. It currently operates three schools to provide special education to children with the learning disabilities associated with brain injury. It also provides the general public with information about brain injury, its symptoms and causes, including lead poisioning.

3 The Center for Science in the Public Interest is a not-for-profit corporation organized under the laws of the District of Columbia and has its main office at 1779 Church Street, N.W., Washington, D. C. Its purpose is to provide scientific information on issues of public importance and to promote adequate goverment safeguards of the public health.

4 Environmental Action Coalition is a not-for-profit corporation organized under the laws of the District of Columbia and has its main office at 1346 Connecticut Avenue, N.W., Washington, D. C. 20036. It has approximately 7,500 supporters. Its purposes are to protect the environment and safeguard human health.

5 Friends of the Earth is a not-for-profit corporation organized under the laws of the State of New York, which has its main office in San Francisco, California, and a branch office at 620 C Street, S. E., Washington, D. C. It has 28,000 members throughout the country. Its purposes are to preserve the human environment and protect human health from pollution.

6 The Highway Action Coalition is composed of approximately 20 membership organizations. It has its main office at Room 731, DuPont Circle Building, Washington, D. C. In addition to the 20 organizations which form the coalition, Highway Action has 3,000 direct supporters. Its purpose is to promote development of sane transportation policies in the United States and to oppose the harmful environmental effects of extensive use of automobiles.

7 The National Welfare Rights Organization (NWRO) is a coalition of 40 state chapters located in 40 different states, including New York, and having a total membership of 16,000 individuals. The New York State chapter has members living in New York City. The national organization has its offices at 1420 N Street, N.W., Washington, D. C. 20005. The purpose of NWRO and its chapters is to protect the rights and interests of poor people, particularly those who are dependent on public assistance. It is particularly interested in preventing lead poisoning from any source. Its members and their children, like all poor people, are victimized by lead poisoning more than the population as a whole, because they frequently live in urban areas where ambient lead, lead-laden dust and lead based paint abound.

B. Defendants

8 Russell Train is the Administrator of the Environmental Protection Agency of the federal government, and, as such, is responsible for the administration of that agency in accordance

with the Clean Air Act, 42 U.S.C. §1857 et seq., and the Administrative Procedure Act, 5 U.S.C. §500 et seq.

9 The U. S. Environmental Protection Agency is the agency of the federal government responsible for administering the Clean Air Act, 42 U.S.C. §1857 et seq.

II. NOTICE.

10 EPA was given appropriate and timely notice of the allegation that it had illegally failed to list lead pursuant to Section 108(a)(1) in compliance with Section 304.

III. THE HEALTH EFFECTS OF AIRBORNE LEAD.

11 EPA has found that:

a. In severe cases, lead poisoning results in death or serious illness, including irreversible brain damage. EPA's position on the Health Implications of Airborne Lead at III 1-3. (Nov. 28, 1973.)

b. Lead exposure insufficient to cause overt symptoms alters vital bodily functions, such as the production of hemoglobin. Id. at III-9 to 10. On the basis of these and other effects, EPA has defined as unacceptable blood lead levels of 40 micrograms per hundred grams of blood or greater (40 ug/100 g). Id. at VII. The effects in children of lead exposure indicated by such unacceptable blood lead levels consist of subtle damage to the nervous system (including hyperactivity and loss of motor control), kidneys, and other systems. Id. at III-5 to 11.

c. Airborne lead has resulted in street dust and dirt in cities, such as Washington, D. C., and Boston, containing up to one percent lead (Id. at II-6), a level which exceeds the concentrations permitted in paint by Federal regulations.

d. Millions of adults and up to 25% of children tested in urban areas are over-exposed to lead. This exposure is caused by a combination of sources of lead including food, water, leaded paint, airborne lead and dust and dirt pervading the metropolitan area. This exposure causes unacceptable blood lead levels in the children, "a small, but significant" portion of all adults, and an important portion of groups of workers, such as policemen and parking attendants, who are highly exposed in their work to an urban atmosphere contaminated with motor vehicle fumes. Id. at VII and Tables.

e. Airborne lead is implicated in clinical lead poisoning. "For a child with pica for paint, the combination of the ingestion of a few chips of paint and an increased intake of lead from contaminated dusts would provide a total lead intake sufficient to cause systematic illness." Id. at VI-6. Since "young children mouth and actually eat things that are not food rather indiscriminately" . . . "dustfall from airborne lead, if swallowed, can make a significant contribution to a small child's total lead intake and thereby contribute to the occurrence of lead poisoning, especially in urban areas." Id. at VI-20 to 21.

f. In New York City, as many as half the children with elevated blood lead levels (between 35 and 44 ug/100 g) could not be associated with living in homes containing peeling leaded paint. Lead contaminated dusts, among other sources, may be "an important contributing factor." Id. at VI-13.

IV. THE REQUIREMENTS OF SECTION 108(a)(1).

12 Lead has an "adverse effect on public health and welfare" and the Administrator has so found "in his judgment" on numerous occasions and at all relevent times. The most recent statement

makes explicit that this finding was within the meaning of Section 108(a)(1)(A). EPA's brief (p. 43) in PPG Industries, Inc. v. EPA, (D. C. Cir. No. 73-2268).

13 Lead "results from numerous or diverse mobile or stationary sources" and the Administrator has so found on numerous occasions and at all relevant times.

14 Lead was not the subject of air criteria issued before the date of enactment of the Clean Air Amendments of 1970.

15 Operating under the Air Quality Act of 1967, the Department of Health, Education, and Welfare (EPA's predecessor in the area of air pollution) stated to Congress that it planned to issue air quality criteria documents for lead. These statements were made in the course of hearings on the Clean Air Act.* In addition, HEW promised environmental groups that it would finalize a criteria document for lead by January 1, 1971. Exhibits 1, 2b, 2c to the Environmental Defense Fund's Supplemental Petition (filed with EPA on November 2, 1971).

V. LEAD IS NOT LISTED

16 On January 7, 1971, EPA produced a draft air quality criteria document for lead. EPA Office of Criteria Development, Airborne Lead.

17 The Administrator did not place lead on the initial list pursuant to Section 108(a)(1) issued within 30 days from enactment has not listed lead subsequently, and has taken no action to

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*Statement of Administrator, Environmental Public Health Service, Public Health Service, HEW, Hearings on S. 3229, 3466, 3546. Before the Subcomm. on Air and Water Pollution of the Sen. Comm. on Public Works, 91st Cong., 2d Sess. 196 (1970); Testimony of Administrator, Environmental Public Health Service, Public Health Service, HEW, Hearings on H.R. 12934, 14960, 15137, 15192, 15848, and 15847, Before the Subcomm. on Public Health and Welfare of the House Commerce Comm., 91st Cong., 1st and 2d Sess's. 293 (1970).

curb lead pollution pursuant to the health protection procedures of Sections 108-110.

18 Internal EPA memoranda indicate that treating lead under the health protection procedures was considered for almost a year after enactment. Mid-1976 is mentioned as the date by which a primary national air standard for lead would have to be attained.

19 The administrator publicly announced his decision not to list lead on December 6, 1973, giving as his sole reason treating a pollutant under the health protection procedures is "discretionary with the Administrator."

20 The Administrator has put only one pollutant, nitrogen oxides, on the initial list, and in the over three years since then, has not added any other pollutant to the list.

VI. IMPACT ON PLAINTIFFS

21 Plaintiffs' members and members of the public generally are exposed to airborne lead.

22 Airborne lead reaches the human body through breathing air and contact with dust and dirt contaminated with settled airborne lead, although, to a smaller extent, airborne lead is present in drinking water and foods.

23 Airborne lead comes from both stationary and mobile sources.

A. Stationary Sources

24 Stationary sources of lead include butare not limited to extraction and processing of lead and other metals, battery factories pigment manufacturing plants, combustion of waste crankcase oil, and incineration of municipal refuse.

25 EPA estimated that in 1970 stationary sources emitted 36 million pounds of lead.

26 In a town near one lead smelter, 80% of the children between one and four years of age had blood lead levels in excess

of 40 mg/100 g.

27 EPA believes that lead emissions from another stationary source is the likely cause of fatalities in horses.

28 EPA has not, to date, put direct limitation on the lead emissions of stationary sources.

B. Mobile Sources

29 Vehicles emitted 350 million pounds of lead from gasoline in 1970, with the biggest concentrations in urban areas.

30 On January 10, 1973, the Administrator published regulations requiring the use of no-lead gasoline in certain model year 1975 and thereafter vehicles to protect their emission control systems pursuant to section 211(c)(1)(B). 38 Fed. Reg. 1258. But, at the same time, he delayed final action on proposed regulations on the lead content of gasoline to be used in the 100 million pre-1975 vehicles in order to protect health, pursuant to section 211(c)(1)(A).

31 A groups of organizations, including some of the Plaintiffs herein, filed a petition for review challenging the delay in the issuance of health protection regulations. NRDC, et al. v. EPA, No. 72-2233 (D. C. Cir.).

32 On October 29, 1973, the Court of Appeals ordered EPA to take final action on these regulations within 30 days.

33 On December 6, 1973, the Administrator promulgated fuel additive regulations for lead under Section 211(c)(1)(A).

34 The regulations will leave from 35 to 40% of the lead in gasoline in 1979, the final year of reductions.

C. Ambient Air Levels for Lead

35 From at least January 7, 1971, to the middle of 1972, EPA believed that an air lead level of 2 ug/m^3 was adequate to protect health.

36 On November 29, 1972, EPA issued its "Position on the Health Effects of Airborne Lead." This document extensively analyzed the research and, on the basis of this analysis, concluded:

> These results cast doubt on the adequacy of the previous position taken by EPA that achievement of a 2ug/m^3 air lead goal would assure a reasonably complete degree of public health protection. This is especially true in view of the possibility (a) that blood lead levels at or above 30 ug/100g in mothers might contribute to similar blood lead levels among their newborn babies and (b) that air lead levels around 2 ug/m^3 may be associated with potentially harmful levels of lead in dust and dirt. On this basis, further air lead reductions below 2 ug/m^3 would seem indicated. Id. at VII-5.

37 Subsequent to the November, 1972, EPA issued "EPA's Position on the Health Implications of Airborne Lead" (November 28, 1973). This and other more recent EPA documents choose not to specify an air lead levels adequate to protect health.

38 EPA's Summary Report on Lead from Stationary Sources (External Draft, May, 1974) presents data from stationary source orientated lead studies for four primary smelter sources in the United States. The following average concentrations from the most impacted sites were as follows:

Southern Solano County, California Study (1970)	3.49 ug/m^3
El Paso, Texas Study (1972)	6.6 ug/m^3
Helena Valley Montana Study (1969)	3.89 ug/m^3
Missouri Study (1972)	5.6 ug/m^3

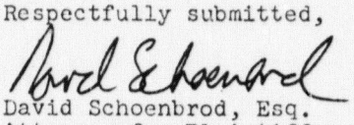
The highest daily average reported was 75 ug/m^3.

39 As originally proposed, EPA's lead in gasoline regulations sought to reduce ambient air concentrations from this source to below 2 ug/m^3 in 1977. As finally promulgated, the regulations

effect the same reductions in 1979. Thus, through 1978, some areas will be exposed to concentrations in excess of 2 ug/m^3.

40 Wherever there are stationary sources of lead, both mobile and stationary sources contribute to ambient air lead levels.

Respectfully submitted,



David Schoenbrod, Esq.
Attorney for Plaintiffs
(Natural Resources Defense Council, Inc.)
15 West 44th Street
New York, NY 10036
(212) 869-0150

November 26, 1974

JSS:n

UNITED STATES DISTRICT COURT
SOUTHERN DISTRICT OF NEW YORK

NATURAL RESOURCES DEFENSE COUNCIL, INC., et al.,

Plaintiffs,

-v-

RUSSELL TRAIN, ADMINISTRATOR U. S. ENVIRONMENTAL PROTECTION AGENCY, et al.,

Defendants.

NOTICE OF MOTION

74 Civ. 4617 (CES)

S I R S :

PLEASE TAKE NOTICE that the defendants, by their attorney Paul J. Curran, United States Attorney for the Southern District of New York, upon the annexed Memorandum of Law, Local Rule 9(g) Statement and Appendices A, B, C, D, E, will move this Court before Honorable Charles E. Stewart, United States District Judge, in Room 2602 of the United States Courthouse, Foley Square, New York, New York at 4:30 P.M. on December 18, 1974, pursuant to Fed.R.Civ.P. 12 and 56 to dismiss the complaint herein, or in the alternative, for summary judgment.

Dated: New York, New York
December 16, 1974

Yours, etc.,

PAUL J. CURRAN
United States Attorney for the
Southern District of New York,
Attorney for Defendants

By: _____
JOHN S. SIFFERT
Assistant United States Attorney
Office and Post Office Address:
United States Courthouse
Foley Square - Room 329
New York, New York 10007
Telephone No.: (212) 791-1973

TO:
DAVID SCHOENBROD, ESQ.
NATURAL RESOURCES
DEFENSE COUNCIL
15 West 44th Street
New York, New York 10036

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UNITED STATES DISTRICT COURT
SOUTHERN DISTRICT OF NEW YORK

NATURAL RESOURCES DEFENSE COUNCIL, INC., ET AL.,

Plaintiffs,

-v-

RUSSELL TRAIN, ADMINISTRATOR U.S. ENVIRONMENTAL PROTECTION AGENCY, ET AL.,

Defendants.

RULE 9(g) STATEMENT

74 Civ. 4617 (CES)

The defendants submit the following statement of material facts as to which there is no dispute in support of defendants' motion for summary judgment and in opposition to plaintiffs' motion for summary judgment.

1. Operating under the Air Quality Act of 1967, the National Air Pollution Control Administration (NAPCA), a Bureau of the Department of Health Education and Welfare (HEW), planned to issue air quality criteria for lead and twenty-three other pollutants. (Hearings before Subcommittee on Air and Water Pollution, p.345.) Under the Air Quality Act, the issuance of air quality criteria and ambience standards was a prerequisite for establishing pollution control standards for all sources except motor vehicles. P.L. 90-148 sections 107, 108.

2. On January 30, 1971, Environmental Protection Agency, (EPA) announced its intention to reduce lead emissions from motor vehicles by controlling lead additives in gasoline pursuant to section 211(c) of the Clean Air Act, 36 Fed. Reg. 1486.

3. The Administrator's announcement on December 6, 1973 that he was acting within his discretion in determining not to list lead under sections 108-110 of the Clean Air Act was a response to a petition by plaintiff NRDC to either issue national ambient air quality standards for lead under sections108-110, or to establish controls on lead in gasoline under section 211 that would reduce lead emissions on the same timetable as would have been achieved under sections 110, 38 Fed. Reg. 33740.

4. Food is a major source of lead content in blood of the general population. Airborne Lead (Ex B) at VIII-2. Other sources include lead in water, air, dust and paint.

5. Lead from stationary sources such as smelters is emitted primarily in the particulate form, usually as an oxide or sulfur compound. EPA has promulgated national ambient air quality standards for particulate matter and for sulfur oxides which require control of existing stationary sources by the states by mid-1975 or at the latest by 1977. 40 CFR sections 50.4-50.7. If a state fails to develop or enforce an adequate control plan, EPA is required to promulgate or enforce standards applicable to specific sources within the state. Sections 110(c)(1), 113; 42 USC sections 1857c-5(c)(1), 1857c-8. For example, EPA has had to propose sulfur oxide control requirements applicable to the Bunker Hill smelter in Kellogg, Idaho. 38 Fed. Reg. 36018 (October 7, 1974).

Federal particulate emissions standards under Section 111 for new or modified stationary sources emitting some lead particulates have been established for incinerators, iron and steel plants, and secondary lead smelters. 40 CFR section 60.52, 39 Fed. Reg. 9308 (March 8, 1974). Federal particulate and sulfur dioxide emission standards have also been proposed for new or modified copper, zinc and lead smelters which emit lead. 39 Fed. Reg. 37040 (October 16, 1974).

EPA is identifying stationary sources of lead not adequately controlled by the foregoing existing or proposed standards and plans to establish new source performance standards controlling lead emissions from those sources. The first candidates for standard setting are lead additive and battery manufacturing plants. Preferred Standards Path Analysis for Lead. Draft, September 14, 1974; Letter from R. Train, Administrator to D. Schoenbrod, dated November 25, 1974 (Ex.E)

The states will be required to control lead emissions from any existing sources of lead subject to federal new source performance standards for lead. Section 111(b), 42 USC Section 1857c-6(b).

6. On December 6, 1973 the Administrator stated in part:

> "After evaluation of the public comment and additional information on this issue, the Administrator determined that it was difficult, if not impossible, to establish a precise level of airborne lead as an acceptable basis for a control strategy. The original health effects analysis was revaluated in view of this finding....

"The lead exposure problem is caused by a combination of sources including food, water, air, leaded paint, and dust. The aggregate contribution of lead from all these sources poses a significant threat to health. However, it is extremely difficult to determine what percentage of the problem each separate environmental factor contributes. Since these are additive sources whose importance varies considerably among individuals it is likewise difficult to determine what impact would be achieved by partial or total reduction of lead from any source."

38 Fed. Reg. Section 3734

7. In other respects, the plaintiffs' Statement of Facts is acceptable to the defendants for purposes of this motion.

Respectfully submitted,

PAUL J. CURRAN
United States Attorney for the
Southern District of New York
Attorney for defendants

By: _____
JOHN S. SIFFERT
Assistant United States Attorney
Office and Post Office Address:
United States Courthouse
Foley Square
Room 329
New York, New York 10007
Telephone: (212) 791-1973

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federal register

THURSDAY, DECEMBER 6, 1973
WASHINGTON, D.C.

Volume 38 ■ Number 234

PART III

THE NATIONAL ARCHIVES OF THE UNITED STATES • 1934 •

ENVIRONMENTAL PROTECTION AGENCY

■

FUEL REGULATIONS

Control of Lead Additives in Gasoline

Appendix A

Title 40—Protection of Environment

CHAPTER I—ENVIRONMENTAL PROTECTION AGENCY

PART 80—REGULATION OF FUELS AND FUEL ADDITIVES

Control of Lead Additives in Gasoline

On February 23, 1972 (37 FR 3882), the Administrator proposed regulations providing for the general availability of lead-free gasoline by July 1, 1974 and a reduction in the lead content of leaded gasoline to 1.25 grams per gallon by 1977. The lead-free gasoline regulations were proposed primarily to ensure the availability of lead-free fuel for use in automobiles designed to meet Federal emission standards with lead-sensitive emission control devices. The Agency recognized that these regulations would also result in a reduction in lead emissions from the new automobile segment of the vehicle population, which would be equipped with those devices. However, based on public health consideration, it was considered necessary, to propose a reduction in the lead content of leaded gasoline as well.

After consideration of the information provided during public hearings and an extended comment period, as well as additional information on the health effects of airborne lead and the adverse effects of lead on emission control devices, the Administrator determined that the two regulations should be dealt with separately. On January 10, 1973, the regulations providing for lead-free gasoline were promulgated, and the regulatory sections providing for a reduction in the lead content of leaded gasoline were reproposed. (38 FR 1255 and 38 FR 1258)

The leaded gasoline regulations were reproposed because the Agency's position on the health effects associated with lead emissions changed substantially. The Administrator had originally proposed the regulations based on the conclusions that airborne lead levels exceeding 2 micrograms per cubic meter were associated with a sufficient risk of adverse physiological effects to endanger public health. After evaluation of the public comment and additional information on this issue, the Administrator determined that it was difficult, if not impossible, to establish a precise level of airborne lead as an acceptable basis for a control strategy. The original health effects analysis was revaluated in view of this finding. The resulting new health position paper concluded that airborne lead can either be directly absorbed through the lungs as people breathe, or can settle out of the air to contaminate dirt which may be consumed by children. Strong evidence existed which supported the view that through these routes airborne lead contributes to excessive lead exposure in urban adults and children. In light of this evidence of health risks, the Administrator concluded that it would be prudent to reduce preventable lead exposure.

The reproposed regulations provided for a reduction in the average lead content of leaded gasolines to 1.25 grams per gallon over a four year period as follows: 2.00 grams per gallon in 1975, 1.70 grams per gallon in 1976, 1.5 grams per gallon in 1977, and 1.25 grams per gallon in 1978. The specified average lead levels referred to the average lead levels of leaded gasoline produced by an individual refinery during any quarter of the specified year.

The final regulations contain a revised lead-reduction schedule based on the Administrator's determination that averaging over all grades of gasoline, including lead-free grades, is preferable to averaging over the leaded grades alone. The schedule has been adjusted to moderate the impact in the early years and to extend it for an additional year. This is discussed in greater detail below. The revised schedule prescribes lower allowable lead content levels, but the overall amount of lead used in gasoline would equal the lead usage expected to result from the reproposed leaded grade reduction schedule in 1979. The reduction schedule under this total pool averaging approach is 1.7 grams per gallon in 1975, 1.4 grams per gallon in 1976, 1.0 grams per gallon in 1978, and 0.5 grams per gallon in 1979. The various averaging alternatives considered by the Administrator are discussed below.

The reproposed reduction schedule was designed to accomplish a 60–65 percent decrease in lead usage from base 1971 by supplementing the projected increasing use beginning in 1974 of lead-free gasoline by new automobiles with catalytic (lead-sensitive) emission control systems. The schedule promulgated below also is designed to achieve the targeted decrease, and generally maintain the reproposed average lead contents for the leaded grades of gasoline.

The Administrator's judgment is that the promulgated reduction schedule is reasonable from the standpoint of protection of health and from the standpoint of economic and technological feasibility. While implementation of this schedule is reducing lead content of gasoline, a joint effort will be made by the Agency and the Department of Health, Education and Welfare to further examine lead emissions from automobile exhausts, to determine whether additional regulation is necessary.

Statutory basis. Section 211(c)(1) of the Clean Air Act authorizes EPA to "control or prohibit the manufacture, introduction into commerce, offering for sale, or sale of any fuel or fuel additive for use in a motor vehicle or motor vehicle engine * * * if any emission products of such fuel or fuel additive will endanger the public health or welfare". The scheduled reduction in the use of lead additives in gasoline to achieve a significant reduction in lead emissions from motor vehicles by 1978 is based on the finding that lead particle emissions from motor vehicles present a significant risk of harm to the health of urban populations, particularly to the health of city children. It is the Administrator's view that the statutory language quoted above does not require a determination that automobile emissions alone create the endangerment on which controls may be based. Rather, the Administrator believes that in providing this authority, the Congress was aware that the public's exposure to harmful substances results from a number of sources which may have varying degrees of susceptibility to control.

Health implications of airborne lead—Introduction. The issue concerning the contribution of automobile lead exhausts to the country's lead exposure problem is complex and controversial. In order to complete a fair assessment of this problem, EPA has made a concentrated effort to obtain and review all the medical and scientific evidence. The Agency has repeatedly requested information and comments from the medical and scientific communities as well as the general public. Since the reproposal of the regulations, information gathered through the comment period on the reproposed regulations, earlier comment periods on the originally proposed regulations, and surveys of relevant studies by EPA personnel have been thoroughly reviewed and evaluated by a task force of EPA medical experts and scientists. A paper entitled "EPA's Position on the Health Implications of Airborne Lead" sets forth in detail the Agency's evaluation that there is a health basis for reducing the use of lead in gasoline. A copy of this paper is available from the Publications Section, Environmental Protection Agency, 401 M Street SW, Room 238W, Washington, D.C. 20460.

General summary of health issue. Environmental lead exposure is a major health problem in this country. A small but significant portion of the urban adult population and up to 25 percent of children in urban areas are over-exposed to lead. The lead exposure problem is caused by a combination of sources including food, water, air, leaded paint, and dust. The aggregate contribution of lead from all these sources poses a significant threat to health. However, it is extremely difficult to determine what percentage of the problem each separate environmental factor contributes. Since these are additive sources whose importance varies considerably among individuals it is likewise difficult to determine what impact would be achieved by partial or total reduction of lead from any source. Should the lead in all sources be reduced, however, it seems clear that the situation would be substantially improved. Leaded gasoline is a source of air and dust lead which can be readily and significantly reduced in comparison to these other sources. It is also one of the few lead sources not yet subject to any controls other than EPA's lead-free gasoline regulations.

Lead from gasoline accounts for approximately 90 percent of airborne lead, total lead additive usage being well over 200,000 tons a year. Lead from stationary sources and deteriorating leaded paint from buildings, combined with lead from gasoline cause high lead levels in dirt and dust. Of these sources, lead from gasoline is the most ubiquitous source of lead found in both the air and the dirt and dust in urban areas. Human exposure to this lead takes place by in-

halation and by ingestion of dirt and dust contaminated by air lead fallout. Since exposure to lead among the general population is widespread, it is reasonable that efforts be made to reduce preventable sources of lead exposure including lead emissions resulting from lead in gasoline.

Many of those disagreeing with the reproposed regulations based their comments on EPA's failure to show sufficient evidence of adverse health effects specifically caused by the use of lead additives in gasoline. While most agree that the combustion of leaded gasoline causes an increase in the amount of lead in the environment, they do not believe that lead in gasoline represents a sufficient endangerment to health or a sufficient risk to the environment to warrant promulgation of controls. The arguments against the position set forth in EPA's reproposed regulations include the following: (1) EPA has failed to show a clear correlation between lead levels in the air and those in the blood of exposed individuals; (2) lead from dust and dirt does not represent a significant threat to body burden of lead; (3) leaded paint is the primary cause of childhood lead poisoning and lead in gasoline does not play an important role in lead poisoning or excessive lead exposure; (4) lead in food and water and not airborne lead are the principal sources of lead to the general population.

A discussion of the four major areas of criticism and a summary of the significant new information received since the regulations were reproposed are provided below.

I. *Is there a correlation between air lead levels and blood lead levels?* A portion of the comments received were critical of EPA's reproposed regulation on the basis that consistently strong correlations have not been found between air lead and blood lead levels. The conclusion expressed by many comments is that except for persons whose occupations bring them in close contact with environmental lead, exposure to airborne lead does not contribute to increased blood lead levels and does not pose a significant threat to health.

These comments cite several studies which did not demonstrate a strong correlation between air lead and blood lead levels. For example, The Seven Cities Study did not show a close correlation between increase in blood lead levels and simultaneous increases in air lead exposures. Blood lead levels were lower among the New York City residents studies than the Philadelphia residents, despite the fact that air lead exposures among the New York residents were actually greater than those in Philadelphia. Also cited as evidence against EPA's position is the observation that despite significant increases in the use of lead in gasoline in recent years there have been no discernible increases in blood lead levels of populations so exposed.

Residential differences in blood leads levels have also not always corresponded to differences in air lead exposures. For example, studies of primitive populations, as well as studies of rural U.S. populations, have shown that the blood lead levels in some of these groups are as high or higher than those of persons living in industrial areas, even though the air lead levels in those rural areas should have been much lower. A comparison between London day and night taxi drivers has also shown no significant differences in blood lead levels but did find differences in exposure to carbon monoxide suggesting that despite the possibility that air lead exposure in the day may have been higher than at night, this was not reflected in blood lead increases. However, differences in smoking intensity, as well as actual differences in air lead exposure between groups, could explain these results and neither were measured.

In summary, a number of comments have criticized EPA's position on the basis that there is not a good correlation between air lead exposure and blood lead levels.

The Agency has weighed against these criticisms studies which have shown that airborne lead does contribute significantly to lead exposure in the general population. For example, using a pilot lead isotope approach, preliminary data show that airborne lead at 2 $\mu g/m^3$ can contribute as much as ⅓ to total lead exposure in man. This result is consistent with data concerning the deposition of lead particles in the pulmonary tract and the absorption of such particles into the blood stream.

An unpublished study in Japan similar to the Seven Cities Study, but which has not yet been completely analyzed, has preliminarily demonstrated that airborne lead exposures below 2 $\mu g/m^3$ affect blood lead levels.

Chamber studies in carefully controlled environments, have shown significant increases in blood lead of men exposed to air lead slightly greater than 3 $\mu g/m^3$.

Differences in the blood lead levels between urban and suburban residents in the same geographic area have been found. When comparable groups with similar lead intakes from other sources besides air were studied, blood leads were consistently higher in urban areas and near highways where air lead concentrations were greatest. Thus while correlations between blood leads and air lead at lower exposure levels are not always good, the evidence indicates that air lead does contribute to general population lead exposure.

Failure to find consistent correlations does not in the Administrator's judgement invalidate the above conclusions. Studies which have come to contrary conclusions have generally failed to take into account the influence of other sources of lead on blood lead levels in people being studied. In the Seven Cities Study, for example, these other sources of lead influencing blood lead levels were not adequately considered in the blood lead-air lead comparisons. EPA has reanalyzed the Seven Cities Study and has found that air lead was a significant, though not the most influential factor affecting blood lead levels. Further, in the Seven Cities Study, urban-suburban differences in blood leads between comparable groups were consistently found which at least in part reflect differences in air lead exposure.

In summary, absorption of air lead does contribute to total lead exposure and when added to lead from other sources such as food and water results in total exposure that is excessive. Thus, the partial removal of lead from the air will help to reduce the degree of excess lead exposure which currently exists among adults and children in the United States.

II. *Does dust lead contribute to lead poisoning in children?* Many comments received by the Agency express the viewpoint that the primary cause of lead poisoning in children is ingestion of lead-based peeling paint. Investigations of cases of clinical lead poisoning in children have repeatedly demonstrated peeling leaded paint as the major source of exposure. Since peeling leaded paint has consistently been observed in the environment of lead poisoned children, many commentors thought it unlikely that lead in dust and dirt could make a significant contribution to this problem. They also point out that lead in dust could be caused by peeling or erosion of leaded paint in or near a home.

One commentor cites X-ray studies of the abdomen among children with lead-poisoning as showing paint chips in the majority of instances. Another argues that differences in blood lead levels between Black and Puerto Rican children could not be explained by exposure to different quantities of lead in dust. Further, studies have shown that animals do not absorb lead from dust as readily as they absorb lead from paint.

Commentors have criticized the Agency for considering that the El Paso Study supports the dustfall hypothesis related to lead in gasoline. In the El Paso Study, children living near a leadsmelter were examined for blood lead levels and for sources of lead in their environment. These results showed that children living nearest the smelter had the highest blood lead levels and that dust lead was a probable major cause. Many commentators, however, considered the El Paso Study applicable only to stationary lead sources and not to lead in gasoline which is different in particle size and chemical composition from smelter-emitted lead.

EPA recognizes the importance of leaded paint as a source of lead exposure for children and that it is the primary cause of clinical lead poisoning. However, based on the evidence available to it, EPA does not believe that leaded paint is the only significant source of lead contributing to excessive lead exposures in children. The Agency's position is that numerous sources contribute to childhood exposure including lead in food, water, air, dust, and dirt as well as paint. Among these sources, contaminated dust and dirt from motor vehicles exhausts are believed to be important exposure routes.

Currently, the contention that lead contamination of dust and dirt by automotive emissions is a significant source of lead exposure is a hypothesis consistent with information provided by a vari-

ety of studies. However, at this time, not all links in the argument have been established beyond dispute and no single study has collectively inter-related all steps in the exposure process to conclusively inter-related all steps in the exposure process to conclusively prove or disprove the hypothesis. Despite the existing uncertainties, comments received from the majority of scientists not affiliated with industrial or environmental groups support the contention that dust is an important source of exposure. This is based on the following evidence:

A. Environmental sampling in a number of cities has demonstrated the ubiquitous presence of lead contaminated dust in urban areas. These measurements were taken inside and outside of buildings including homes and schools. Dust lead measurements outside homes commonly ranged from 0.1 to 0.5 percent lead by weight. Measurements well in excess of 0.5 percent have also been recorded. Inside homes, samples were found to contain lead contents ranging from 0.05 to 0.2 percent and in some instances as high as 0.5 percent. Current Federal regulations have already established that lead concentrations in paint in excess of 0.5 percent represent a definite hazard to children and serious consideration is being given to reducing the allowable level to 0.06 percent. In testimony before the United States Senate, Dr. Merlin DuVal, at the time Assistant Secretary for Health and Scientific Affairs at HEW, commented on an appropriate safe level for lead in paint:

> Based on information now available to us, we are satisfied that it is technologically feasible, and desirable from a health viewpoint to move toward the .06 percent standard recommended by the American Academy of Pediatrics.

B. As was stated above, high lead concentrations in dust are prevalent in urban areas. It is not clear in all instances, which sources are contributing most to this contamination. Comments received by the agency point out that high lead levels in some cases may be caused by the chipping or peeling of leaded paint from interior and exterior surfaces. EPA agrees that this is true. In other cases, the lead dust content is clearly the result of lead emission from stationary sources such as smelters. However, EPA believes an important and the most ubiquitous source of lead in dust is the exhaust of automobiles using leaded gasoline. Annually, over 200,000 tons of lead are used as additives in gasoline. The vast majority of this lead is emitted into the environment. Although significant amounts of lead remain airborne for extended periods of time, evidence indicates that a large quantity of the exhaust lead rapidly settles to the ground within several hundred feet of the source. Measurements of lead in dust and soil further indicate that lead content decreases with increased distance from the roadway. It has also been found that dust lead levels in homes near heavily traveled roadways are significantly higher than in comparable homes located along side streets. It should be noted that the majority of studies reporting high levels of lead in dust and dirt did not associate sources of peeling leaded paint or stationary lead sources with the lead dust measurements. Accordingly, the Agency believes that in most circumstances lead from automobile exhaust is the primary source of lead in dust and soil in urban areas.

C. The general environment of urban children commonly includes dirt and dust contaminated with lead. A large percentage of children, especially between the ages of one and three years, are known to ingest non-food objects in their mouths. It has been demonstrated that children living in high dust lead environments have greater quantities of lead on their hands than children living in less contaminated environments. The existence of leaded dust on the hands of urban children has been highlighted by the common occurrence of inadvertent lead contamination of finger prick blood lead specimens taken from these children.

D. Children who ingest leaded dust and dirt can be expected to absorb some of the lead into their bodies. Though it is difficult to determine the precise amount of lead that would be absorbed, animal experiments suggest that appreciable quantities of this lead, whether from smelters, paint or gasoline exhaust, are absorbed. Further, it has also been shown that at least some children residing in environments heavily contaminated by leaded dust and dirt absorb enough to suffer from subclinical and even clinical effects of lead overexposure. This was particularly true in the case of El Paso, mentioned above. Though the lead source was a smelter, animal studies indicate that lead in dust due to leaded gasoline would be absorbed in quantities comparable to that emitted by the smelter. Another study from Charleston, South Carolina indicates that children residing in homes near high soil lead concentrations had a greater frequency of lead poisoning than children residing in less contaminated areas. This study suggests that lead from soil was absorbed, although it is not clear what sources were primarily responsible for those high soil lead levels. It should be further noted that instances such as those above, coupled with known high levels of lead in dirt and dust, indicate that children could easily ingest enough lead by this route to be significant.

E. Various studies indicate that cases of lead poisoning and significant overexposure are not always associated with urban home environments in which sources of peeling or chipping leaded paint were observed. These studies include children residing primarily in inner city areas. Admittedly children may be exposed to peeling or chipping leaded paint in environments away from their own homes. However, since several recent studies indicate that up to 50 percent of children with excessive lead exposure are known to not reside in homes where peeling lead based paint can be found, it is unlikely that peeling paint exposure away from the homes accounts totally for this difference. Furthermore, extension of blood lead screening programs outside of slum areas indicates that the lead exposure problem is found in children residing in higher income areas where peeling paint is not frequent and exposure to this source away from the home is less likely. In conjunction with these findings, residence near roadways have been found to contain higher quantities of lead than those measured away from the road. Findings such as these indicate that in some circumstances dust lead is an important factor and at times may be the primary factor contributing to excessive lead exposure associated with subclinical if not clinical effects.

F. Clinical symptoms resulting from very high lead exposure in children are known to be associated with permanent neurologic damage. It has also long been suspected, but not proven beyond doubt, that lead exposures below those sufficient to cause clinical symptoms in children are also harmful. In particular it has been observed that physiologically significant biochemical changes occur in children with excessive exposures below clinical toxicity and it has been proposed that these changes are reflective of subclinical changes that precede overt disease. Recently available scientific information, though far from completely resolving this issue, supports the view that adverse effects due to lead in children are not confined only to situations in which overt clinical symptoms of lead poisoning occur. Included in these findings are increased subtle neurological impairments among children more highly exposed to lead below levels known to cause clinical disease.

III. *Will a reduction of lead in gasoline reduce the incidence of clinical lead poisoning in children?* Ingestion of peeling paint has long been recognized as the primary cause of clinical lead poisoning in children. This position has been expressed in many comments received by the Agency including those from several noted authorities in the field of lead poisoning. For this reason, numerous comments have questioned the need to reduce lead in gasoline on the basis that this action would have little if any impact on reducing the incidence of clinical lead poisoning in children.

While EPA recognizes the importance of leaded paint as a source of lead for children and has supported governmental efforts to reduce this risk, the findings of several studies suggest that lead poisoning can develop in the absence of significant sources of leaded paint. Though this possibility does not affirm that reducing lead in gasoline will reduce the incidence of lead poisoning in children it indicates that lead in gasoline may, in conjunction with other nonpaint sources, contribute to the development of lead poisoning. Whatever the impact this reduction may have upon clinical lead poisoning, the action will significantly reduce lead exposure among children.

EPA is also concerned about the probability that children exposed to lead at

levels below those associated with clinical poisoning are also being adversely affected. Several effects identified as subclinical lead effects include impairment of fine motor functions, and altered behavior.

It is noteworthy that in a significantly large percentage of excessive lead exposure cases (up to 50 percent in some instances) peeling lead based paint in the home cannot be identified as a source of the exposure. Thus, while leaded paint is recognized as the major cause of childhood lead poisoning, it is not clear that leaded paint is singly responsible for the large degree of excess childhood lead exposure in this country.

IV. *Excess lead exposure among the general population could result from a combination of lead sources, not one of which by itself is sufficient to be a problem. Under these circumstances, would it not be preferable to formulate a control strategy based upon reducing lead levels among those sources that contribute the most to this total exposure?* It is generally agreed that food is the major source of lead to the general population. A World Health Organization expert committee reports that according to the results of total diet studies in industrialized countries, the total intake of lead from food generally ranges from 200–300 ug per person per day. WHO further states that based upon available data, these levels are similar to those found in the past 30–40 years and that no upward trend in lead levels in food is evident.

This information suggests that the level of lead in food has remained relatively constant in recent times. Though lead in food would certainly contribute to total lead exposure for the general population, lead in food is probably not the source that is most readily reduced in the event that total exposure to lead is excessive. According to WHO, "Any increase in the amount of lead derived from drinking water or inhaled from the atmosphere will reduce the amount that can be tolerated in food. The lead in air is probably the contribution that is most accessible to action for reducing the total body burden of lead, especially where this fraction is large compared with that absorbed from food."

V. *What new information has become available since reproposal of the regulation and as a result of the additional comment period?* The majority of comments addressed the evidence presented by EPA in support of its proposed regulation and did not introduce new evidence. The number of comments received were approximately evenly divided between those in favor and those against. The bulk of comments critical of EPA's health position was submitted by industry or industry affiliated scientists. Independent scientists who commented, not affiliated directly with the industry or environmental groups, were in favor of the regulation by approximately 2/1. Most favorable comments, though often from scientists knowledgeable in the field of lead, provided testimonial support rather than new evidence. Most new data that either was presented in comments or which subsequently became available to EPA does support the need to reduce lead emissions from automobiles. Among these latest data are the following:

(1) Studies of subclinical lead effects in children continue to suggest that fine motor function and behavior are affected. Though this issue is not completely resolved, the new data emphasize the potential subclinical risk.

(2) It has been reaffirmed that high dust lead levels, up to 1% lead content, have been found in children's play areas, inside schools and in homes.

(3) New evidence reaffirms that high dust lead levels can be caused by leaded gasoline. A recent study in Rochester, New York, demonstrates that high dust lead levels in homes are not always associated with peeling paint and that house dust lead levels are higher in urban than suburban homes. A study in Vermont has shown that higher concentrations of lead in house dust are found in homes located near busy roads compared to homes on sidestreets. This latter point is consistent with the previously known fact that air lead fallout decreases with increased distance from roadways. A study by EPA in New York City indicates that higher household dust and soil lead levels are found in areas with greater dust lead fallout from the air as compared to areas with little lead fallout.

(4) Young children living in homes with high dust lead contents have been found to have more lead on their hands than children in homes with low dust lead content. This finding provides an important link in the dust fall lead hypothesis. The finding is consistent with observations that finger prick blood-lead specimen taken from children are routinely contaminated by lead that is present on the fingers.

(5) Studies continue to indicate that a high degree of exposure to environmental lead is not confined to inner city areas. Cases of over-exposure continue to be reported from areas in which leaded paint would not be expected to be the predominant factor.

(6) Studies from Newark, New Jersey, observed that the frequency of lead poisoning and undue lead exposure is doubled among childern living close to major roadways compared to children living farther away.

Other means of achieving lead reductions. Before prescribing regulations based on public health consideration, the Administrator must consider "other technological or economically feasible means of achieving emission standards under section 202." Thus, if EPA determined that a reduction of lead emissions from motor vehicles is necessary for protection of public health or welfare, the feasibility of achieving such a reduction under section 202 (new motor vehicle emission standards) must be considered.

The primary alternative to the use of lead additive regulations to achieve reduction in lead emissions would be to impose a lead emissions standard which would result in the installation of "lead-traps" on motor vehicles. The possibilities of incorporating this alternative, however, are limited by the existing legal and technical realities.

EPA does have the authority to impose a lead emissions standard on new vehicles which would result in the use of lead traps. The earliest that such a regulation could be imposed, however, would be the 1976 model year. Most motor vehicle manufacturers are expected to use lead sensitive emissions control systems to meet the Federal emissions standards which are applicable to new vehicles in 1976. Lead traps cannot adequately protect these systems because they are not capable of trapping all of the lead emitted. Lead-free gasoline will be required in most new vehicles based on the information now before the Agency. See Aerospace Report, PB–205–981, available from National Technical Information Service, 5285 Port Royal Road, Springfield, Virginia 22151. Accordingly, the use of lead traps is relevant principally with regard to in-use vehicles. EPA realizes that lead-tolerant emission control systems may be used on a progressively greater number of new vehicles in the future. However, many of the new technology lead tolerant control systems are expected to operate on low octane gasoline which may not require lead additives. Nevertheless, the Agency is continuing to study the feasibility of using lead traps on new vehicles in the future.

The Clean Air Act does not authorize EPA to establish national emission standards on in-use vehicles. Since lead traps cannot be used successfully on the vast majority of new vehicles and the Agency is legally incapable of requiring them on all in-use vehicles, the use of lead traps is really not a feasible alternative at this time in the Administrator's judgment.

Despite the legal authority obstacle EPA has examined the technological capabilities and costs of lead traps and has determined the regulation of lead additive use is the preferable method of controlling lead emissions.

Other emissions. Concern has been expressed that the control of lead additives may result in the use of other gasoline components or additives which may also have an adverse impact on health. EPA has evaluated the potential use of other additives or greater percentages of certain gasoline components in conjunction with the lower lead levels. This evaluation has been performed in recognition of the Agency's responsibility to assess the environmental consequences of its actions. (See Judge Leventhal's opinion in Portland Cement V. Ruckelshaus, 5 ERC 1593, 1599, U.S. App. D.C. (1973).)

Lead additives are used as efficient octane boosters in gasoline. If the use of lead is restricted, the refiner must use greater quantities of blending stocks with high aromatic hydrocarbon concentrations, or substitute anti-knock additives, to increase gasoline octane levels. Consequently, the Administrator has considered the effects of increased aromatic hydrocarbon content of gasoline or the use of manganese additives on emis-

sions from the general motor vehicle population and the effects of these emissions on health. EPA has also considered the impact of the regulations on particulate emissions.

A. *Impact due to increased use of aromatics.* The implementation of the promulgated and reproposed lead regulations is projected to result in a 5 to 7 percent increase in the average aromatics content of gasoline. Concern has been expressed that this increase will cause a complementary increase in the reactivity of automobile exhaust and in the quantity of polynuclear aromatic emissions from the motor vehicle population. EPA has determined that neither the reactivity of automobile exhaust or the emissions of polynuclear aromatics will increase above current levels due to the lead regulations.

Emissions reactivity. Gasoline is composed of three general types of hydrocarbon: aromatics, olefins, and paraffins. Aromatics and olefins are highly reactive and facilitate the formation of photochemical smog. Assuming no hydrocarbon emission controls, aromatics emissions are linearly related to the aromatic content of gasoline. Olefin emissions are directly related to the olefin and paraffin content of gasoline. An increase in the aromatic content of gasoline is accompanied by a decrease in the paraffin and olefin content. Consequently, reactive aromatic emissions increases resulting from an increase in the aromatic content of gasoline are generally offset by a decrease in the reactive olefin emissions due to a complementary reduction in the olefin and paraffin content of the specified gasoline. Accordingly, the increase in the aromatics content in gasoline will not have a significant impact on automobile emissions reactivity.

The lack of increase in exhaust reactivity due to increased use of aromatics has been verified in smog chamber studies completed by the Bureau of Mines as well as EPA. It should also be noted that aromatic emissions from the automobile population will continue to decrease as vehicles with increasingly stringent hydrocarbon emission control systems replace older uncontrolled vehicles on the road.

A detailed analysis estimating exhaust reactivity and the effect of the EPA fuel regulations has been conducted and reported by Dr. A. P. Altshuller in "Effects of Reduced Use of Lead in Gasoline on Vehicle Emissions and Photochemical Reactivity," February, 1972. This paper is available from the Environmental Protection Agency's Office of Public Affairs, Publications Section, Room 238 W, 401 M Street, SW., Washington, D.C. 20460.

Polynuclear aromatic emissions. Polynuclear aromatic hydrocarbons (PNA) are carcinogenic and are primarily caused by hydrocarbon emissions from stationary sources such as petroleum refineries and coke ovens. Currently automobile emissions account for less than 2 percent of total PNA emissions.

Polynuclear aromatic emissions from the general automobile population have been steadily declining since the introduction of hydrocarbon emission controls in 1968. Due to the continued attrition of older uncontrolled vehicles from the road and the introduction of new vehicles with stringent hydrocarbon controls, PNA emissions should be reduced by more than 75 percent from current levels by 1980. This assumes the implementation of both the promulgated lead-free and reproposed low-lead regulations will have a very slight impact on the rate of decrease in PNA emissions. According to a recent EPA analysis, PNA emissions will be reduced by 78 percent by 1980 assuming the implementation of the 1976 hydrocarbon emission standards. If the lead regulations are implemented, PNA emissions will decrease by 76 percent. (An analysis of this problem is contained in a paper entitled "Lead in Gasoline, Impact of Removal on Current and Future Automotive Emissions".) EPA concludes that the current use of lead additives endangers the public health to a greater degree than this difference of 2 percent in the rate of decrease of PNA emissions.

This relative endangerment judgment is based upon the following line of reasoning. Lead additive emissions from automobiles have been determined to pose a sufficient endangerment to health to warrant regulatory action. Mobile sources contribute less than 2 percent of the total polynuclear aromatics emissions. Implementation of the Federal emission standards without the lead additive emissions will result in an approximately 78 percent decrease in polynuclear aromatic emissions from current levels of automobile emissions. Implementation of the lead regulations will slow the rate of emissions decrease by about 2 percent. Assuming automobiles account for the same relative contribution of aromatics in 1980, implementation of the emission standards with the regulations as compared to without the regulations would only cause a 0.04 percent difference in reduction rate in total PNA emissions. In view of the continual decline in PNA emissions and any associated health risk, from stationary sources through particulate controls and from mobile sources through hydrocarbon controls, the health implication of the slight difference in PNA emissions due to the lead regulations is considered negligible.

Although the indication is that the lead regulations will not produce an aromatics or a PNA emission problem, EPA nevertheless has the authority to regulate the aromatic content of gasoline should such action become necessary.

B. *Particulate emissions from unleaded fuel.* Exhaust particulate resulting from the use of leaded and lead-free gasoline has been extensively examined. The examination concluded that since lead additives account for a major portion of exhaust particulates, the use of fuel without lead additives substantially decreases particulates emissions. This conclusion is true for vehicles equipped with emission control devices as well as uncontrolled automobiles.

C. *Use of substitute anti-knock additives.* Various anti-knock additives have been developed, but as is explained in the paper, "Lead in Gasoline" referred to above, the effectiveness of almost all of these additives is severely limited. Manganese is the only fuel additive besides lead which is now recognized as being a cost effective octane booster. While manganese additives are not currently in widespread use in gasoline, manganese may be used as a partial replacement for lead in gasoline. EPA has been examining the impact the use of manganese additives might have on control devices and on the public health.

One automobile manufacturer has recently completed tests using fuel containing 0.25 grams per gallon manganese in vehicles equipped with catalytic emission control systems. While no chemical poisoning was observed, a very high back pressure developed after several thousand miles. This back pressure was due to manganese oxides plugging the catalyst. Apparently, manganese oxides, unlike lead halides, are nonvolatile and physically destroy catalyst functioning by plugging. The 0.25 grams per gallon manganese is above the levels that would be used in fuel by only a factor of two. Accordingly, the plugging problem would eventually occur if manganese is used in lead-free gasoline. Furthermore, deterioration of catalyst performance would occur soon after an individual began using gasoline containing manganese additives. At the present time, the auto manufacturers have not requested that manganese additives be controlled. This may reflect the industry's expectation that manganese additives will not be used in lead-free gasoline. If it is used, EPA would have to consider regulating manganese additives under the authority of section 211(c)(1)(B) of the Clean Air Act.

Occupational experience indicates that airborne manganese at sufficiently high levels of exposure can cause damage to the central nervous system with symptoms similar to that in Parkinson's disease, and can cause manganese pneumonia. Available evidence indicates that dosages required to produce these adverse effects are several orders of magnitude above those that would be present in the ambient air as a result of even the widespread use of manganese as a gasoline additive. Thus, while there presently appears to be a reasonable margin of safety with use of manganese in gasoline, the health implications of this use require continued study. An EPA position paper on manganese is currently being prepared which will be available in the near future. This document will be based upon a comprehensive review of the information available on manganese directed by the National Academy of Sciences.

If regulation of manganese in gasoline for health reasons is found to be necessary, EPA has authority to do so under the Clean Air Act. Though the Agency does not currently have enough evidence to definitely say that manganese in gasoline would pose a threat to health, EPA would not favor the use of manganese in

gasoline until additional studies are completed. However, at this time, the use of manganese additives is judged not to pose as significant a risk to health as that from lead additives.

Cost and energy impacts. Recently EPA has worked with Bonner and Moore Associates to complete a study based on updated information of the impacts associated with the reproposed leaded grade regulations. This study separates the various costs according to two assumptions concerning the portions of the vehicle population which will use lead-free gasolines. The first case assumed all motor vehicles manufactured after 1975 will be equipped with lead sensitive catalytic emission control systems and will thus need lead-free gasoline. The second case assumed an ever increasing portion of the vehicles produced during model years after 1975 will be equipped with emission control systems capable of tolerating leaded gasoline. The second case assumed that by the 1985 model year, all new vehicles will have emissions control systems which can tolerate lead.

Based on this new data, EPA has calculated the annual consumer costs attributable to the low-lead regulations. This calculation includes the increased costs of raw stocks, as well as operating and production costs at the refinery. During 1980, capital investment in the refinery industry is predicted to be roughly $1.5 billion. The low-lead regulation will force the industry to invest an additional $82 million. If a lead-tolerant technology is gradually phased in and thus more leaded gasoline is used, the incremental investment impact of the low-lead regulation will be $113 million. This figure will increase the cost of producing gasoline by less than .1¢ per gallon.

Recently, much concern has been expressed about the potential impact lead regulations would have on the nation's crude oil supply. The low-lead regulations will not go into effect until 1975 and will have a minimal impact on crude oil requirements during this decade. Modeling studies completed by Bonner and Moore Associates demonstrated no positive impact in either 1975 or 1977 on crude usage. If it is assumed that additional leaded gasoline is required to fuel new vehicles equipped with lead tolerant emission control systems which might be partially phased in between 1976 and the end of the decade the impact represents less than a .4 percent increase in crude usage by 1980. If one assumes catalysts are used on all future model vehicles and consequently the quantity of leaded gasoline produced continues to decline, the low-lead regulations never have a significant impact on crude requirements.

It is instructive to compare these numbers with the energy impact of air conditioners in automobiles. Air conditioners have been estimated to have a 13 percent impact on fuel economy. In 1980 if 75 percent of the automobiles are equipped with air conditioners, the impact on crude oil requirements will be approximately 800 thousand barrels per day or roughly 4.4 percent of the nation's needs.

Averaging strategy. The lead regulations proposed on January 10, 1973, would permit each refinery (not company) to average its lead usage over quarterly production of leaded gasoline so long as the average lead content per gallon did not exceed the applicable standard. Leaded pool averaging was proposed for comment based upon the determination that this approach afforded optimum refining flexibility consistent with attainment of the Agency's goal of 60–65 percent reduction in lead usage.

In light of additional information and views received during the comment period, EPA has reviewed the merits of two alternatives to leaded pool averaging. These are (1) a system of total pool averaging, permitting a refinery to average its lead usage over all grades of gasoline produced including the unleaded grade, and (2) permitting each refinery a choice between leaded pool averaging and total pool averaging. Analysis of the impacts and practicalities of the alternative averaging approaches has led EPA to conclude that total pool averaging should be adopted.

Comparing the effects of leaded pool averaging and total pool averaging shows that refiners who market two grades of gasoline, one leaded and one unleaded grade, are significantly penalized by leaded pool averaging. Because two grade marketers are unable to count production of unleaded gasoline in computing the average, a leaded pool standard exerts pressure to market three grades of gasoline, including two leaded grades, to maximize allowable lead usage. Leaded pool averaging similarly tends to penalize three grade marketers who produce more than the industry average proportion of unleaded gasoline. It benefits refiners who produce little or no unleaded gasoline.

Total pool averaging is not expected to induce three-grade marketers to opt for two grades, but does not tend to discourage production of unleaded gasoline. A total pool standard permits each refiner to use the same amount of lead for equivalent gasoline production and is more neutral in its effect upon industry marketing decisions.

The alternative of allowing each refinery a choice between leaded pool and total pool averaging would permit each refinery to choose the system that maximizes lead usage. The price of this flexibility is that lead reduction goals would not be achieved. It is not possible under an option system to predict what reductions in lead usage would be achieved under the regulations. The reductions achievable under an option system would depend on the mix of leaded and unleaded gasoline sales, the sales volumes, and the marketing plans of all gasoline refiners. The option alternative does not permit reasonable estimates of the reductions in lead usage attainable under any given pair of standards.

A majority of the refiners who commented on the regulations recommended that total pool averaging be adopted. The Administrator finds that total pool averaging is in fact the fairest workable mechanism for accomplishing the necessary reduction in lead usage.

Computation of total pool standard. The promulgation of a total pool average standard requires that the reproposed leaded pool standard be adjusted to take account of projected sales of all gasoline. The method of computation is to multiply the numerical leaded pool standard by the percentage of leaded gasoline sales estimated for the particular year. For example, 2 grams per gallon × the percentage of 1975 sales of leaded gasoline = the total pool standard for 1975.

Future sales of unleaded and leaded gasoline cannot be predicted with complete assurance. Actual sales of unleaded gasoline will depend upon the number of vehicles requiring it to meet emission standards, the extent to which owners of vehicles not requiring unleaded gasoline will buy it, and the projected miles driven and fuel consumption of vehicles in the various model year classes.

A study entitled "Alternative Proposals for the Regulation of Lead Additives in Gasoline" prepared for EPA by the firm of Turner, Mason, and Solomon in June, 1972, sets forth estimates based on different assumptions affecting sales of leaded and unleaded gasoline. The estimates selected by EPA as most consistent with present trends in unleaded gasoline sales are provided in case I of the Turner, Mason and Solomon Report. Case I assumes no extension of the 1975 standards, eliminating the need for unleaded gasoline, but that owners of pre-1975 model year motor vehicles will purchase little or no unleaded gasoline.

EPA recognizes that the assumption that owners of pre-1975 vehicles will purchase little or no unleaded gasoline results in conservative estimates of unleaded gasoline sales, but this assumption is offset by the fact that not all 1975 vehicles will require unleaded gasoline.

Using the Case I estimates of future sales of leaded and unleaded gasoline, the conversion of the proposed leaded pool standard to a total pool standard is as follows:

Year	Leaded pool std.	Percent of sales unleaded	Total pool std.
1975	2.0	82.2/17.8	1.644=1.6
1976	1.7	69.8/30.2	1.186=1.2
1977	1.5	59.5/40.5	.89 = .9
1978	1.25	50.9/49.1	.63 = .6
1979	1.25	43.6/56.7	.54 = .5

The promulgated reduction schedule is derived from the table above, but the schedule has been adjusted to moderate the economic and technological impacts of the regulations during the period over which the reductions would be accomplished. To achieve the targeted 60–65% reduction in lead usage requires that the schedule be extended to include 1979. The total pool standard corresponding to the proposed leaded pool standard for 1979 is .5 gram per gallon. As stated above, the schedule promulgated is as follows:

January 1, 1975	1.7 grams per gallon.
January 1, 1976	1.4 grams per gallon.
January 1, 1977	1.0 grams per gallon.
January 1, 1978	.8 grams per gallon.
January 1, 1979	.5 grams per gallon.

This reduction schedule will achieve the 60–65 percent reduction in lead usage and emissions as planned and will also assure that industry's lead usage under total pool standards is approximately the same as the lead usage projected under the leaded pool standards previously proposed.

The standard will have to be evaluated in 1978 to determine what further reductions in the lead standard, if any, are necessary to maintain lead emissions at the desired level. Presumably no further reductions will be required if unleaded gasoline remains the fuel required for new motor vehicles. If unleaded gasoline is no longer required for new vehicles, the 1978 standard will be reexamined in light of increasing gasoline demand.

Combining refineries for purposes of averaging. Two refiners have requested that the regulations be changed to authorize EPA to approve combinations of refineries for purposes of computing the average instead of requiring averaging at each refinery. This approval would be requested to enable a company to concentrate production of leaded or unleaded gasoline at particular refineries.

EPA proposed averaging at each refinery instead of each company in order to mitigate any regional variation in lead emissions due to averaging. Regional variation could result from the mix of gasoline grades sold in a particular market if a company used its lead allotment mainly in one grade or from a company's decision to produce high-lead gasoline at an old southeast refinery and low-lead gasoline at a newer west coast facility, each serving different markets. Requiring lead levels to be moderated at each source is a reasonable effective means of minimizing variation in the area where the gasoline is actually sold.

Although one company has suggested that the location of the refineries in a particular EPA region might serve as a basis for approving combinations, this criterion does not provide assurance that the areas served by those refineries and other refineries would not be subject to variation in lead emissions. There is no necessary correlation between the location of the refineries and their service areas. The Administrator has concluded that there are no workable criteria for assessing the impact of combinations for purposes of averaging and that the refiners' desire for added flexibility in lead usage cannot be accommodated without compromising the objective of minimal variation in reduction in lead emissions in all parts of the country.

Averaging period. Many refiners requested an annual or semiannual averaging period instead of the quarterly period proposed. A longer averaging period would accommodate seasonal variations in lead usage. Because high volatility, high octane blending stocks are used in the winter season to facilitate cold starts, less lead is needed in winter blends. The refiners would like to be free to put more lead in summer blends, and a longer averaging period would make this possible.

The summer season is also the period of maximum exposure to airborne and dustborne lead for both children and adults. For this reason, EPA is unable to agree to the change proposed in the averaging period.

Small refiners. The reproposed regulations provided for a one-year delay of the requirement to comply with the lead reduction schedule for small refiners, as defined in § 80.20(b), in recognition of special lead-time problems faced by this group. EPA has reviewed the lead-time requirements of the small business refiners with particular reference to the effect on lead-time, if any, of the change to a standard based on total pool averaging. The Agency recognizes that under the reproposed leaded pool standard, refiners producing little or no unleaded gasoline received the benefit of a higher average lead level per gallon of leaded gasoline. Some small refiners fall into this category, and would have been able to use more lead under a leaded pool standard taking account of production of unleaded gasoline by other refiners.

EPA's evaluation of the small refiners' situation has led to the conclusion that these refiners require additional lead-time for compliance beyond the one year deferment previously proposed. This appears to be the case regardless of the averaging strategy adopted. Industry and consultants' estimates of time required by small refiners to plan, finance, and construct upgraded refining facilities range from two to three years from the date of promulgation of final standards. Accordingly, the Administrator has determined that it is reasonable and necessary to defer the requirement for compliance by small refiners until January 1, 1977. On this date, small refiners are required to comply with the 1977 standard.

Review of lead reduction program. In the January 10, 1973, reproposal of the regulations, the Administrator stated his intention to reduce the lead content in gasoline as much as possible, giving consideration (a) to the degree of reduction achieved by introduction of unleaded gasoline and (b) evidence on the feasibility of reducing lead from other environmental sources. It is too early to state whether unleaded gasoline sales will expand steadily through the seventies. Studies of potential reduction in lead from other sources are in progress. Accordingly, the Administrator has determined that it would be premature to announce a decision on the need for further reductions in lead in gasoline. EPA will review progress under the regulation as well as additional studies every three years, beginning in 1977. This review will afford a firmer basis for a decision on whether further action is necessary to regulate lead in gasoline to protect public health and welfare.

Reporting by lead additive manufacturers. The January 10, 1973 reproposal included a requirement that lead additive manufacturers would report quarterly to the Administrator on their shipments of lead to each refinery. No comments were received on this proposal, which is promulgated below as proposed. The basis for the requirement—that it is determined to be necessary for verification of lead additive usage reports by refineries—has not changed.

Prevention of violations by refiners. As a complementary measure to the January 10, 1973 promulgation of a strict liability provision in § 80.23 applicable to refiners, the Agency on that date proposed a provision specifying that it is the refiner's duty to prevent violations of § 80.22(a). Two refiners and one petroleum trade association commented that practical and legal considerations made the regulation unreasonable, particularly as regards the requirement on permitting violations. One other refiner commented that if the requirement was to be adopted, it should provide for a showing by the refiner that he in fact did not cause or permit a given violation.

The issue of vicarious liability under § 80.23 is now in litigation and the Agency is engaged in negotiations with refiners which may lead to revision of the provision. Accordingly, no action is being taken on the proposed § 80.20(c), but it is not being withdrawn.

Control of lead under Title I. One commentator has contended that the Clean Air Act requires the Administrator to establish a national ambient air quality standard for lead under Title I or, at least, to impose controls under § 211 that would achieve results which would be as protective of health on as expeditious a timetable as could have been achieved under Title I. The commentator, an environmental group, concludes that the reproposed lead reduction schedule would achieve "far less effective and timely results than action under Title I because the reproposed schedule is so weak," and petitioned EPA for the issuance of national ambient air quality standards for lead.

It is clear from Agency actions to date that the Administrator has chosen to regulate lead emissions under section 211 of the Act. No action on lead under Title I is currently planned.

It is the Administrator's judgment that he may regulate a substance under section 211 without necessarily tailoring his action to what could have been accomplished under sections 108, 109, and 110, since section 211 is a co-equal grant of regulatory authority. The determination whether to issue a criteria document for a substance and thereby set Title I actions in motion is discretionary with the Administrator. Section 108 expressly recognizes this, inasmuch as it required the Administrator to list for action under Title I only those air pollutants "for which air quality criteria had not been issued before the date of enactment of the Clean Air Amendments of 1970, but for which he plans to issue air quality criteria. (emphasis added) This falls considerably short of a statutory directive to issue criteria for lead.

and may be contrasted readily with the requirements of section 202(b) of the Act specifically identifying carbon monoxide, hydrocarbons, and oxides of nitrogen for regulatory action. While, as the commentator points out, language in the Senate Report on its version of the 1970 Clean Air Act amendments stated that the bill would require issuance of a criteria document for lead, this must be construed as only a statement of the Committee's preference, since no such requirement appeared either in the language of the Senate or the conferees' bill.

The regulations promulgated below shall be effective on January 7, 1973.

(42 U.S.C. 1857f-6c, 1857g(a))

Dated: November 28, 1973.

JOHN QUARLES,
Acting Administrator,
Environmental Protection Agency.

Part 80 of Chapter I, Title 40 of the Code of Federal Regulations is amended as follows:

1. In § 80.1, the second sentence is revised to read as follows:

§ 80.1 Scope.

* * * These regulations are based upon a determination by the Administrator that the emission product of a fuel or additive will endanger the public health, or will impair to a significant degree the performance of a motor vehicle emission control device in general use or which the Administrator finds has been developed to a point where in a reasonable time it would be in general use were such regulations promulgated; and certain other findings specified by the Act.

2. In § 80.2, a new paragraph (m) is added as follows:

§ 80.2 Definitions.

* * * * *

(m) "Lead additive manufacturer" means any person who produces a lead additive or sells a lead additive under his own name.

3. A new § 80.20 is added as follows:

§ 80.20 Controls applicable to gasoline refiners.

(a)(1) In the manufacture of gasoline at any refinery, no gasoline refiner shall exceed the average lead content per gallon specified below for each 3-month period (January through March, April through June, July through September, October through December):

(i) 1.7 grams of lead per gallon, after January 1, 1975;

(ii) 1.4 grams of lead per gallon, after January 1, 1976;

(iii) 1.0 grams of lead per gallon, after January 1, 1977;

(iv) 0.8 grams of lead per gallon, after January 1, 1978;

(v) 0.5 grams of lead per gallon, after January 1, 1979.

(2) For each 3-month period (January through March, April through June, July through September, October through December) the average lead content per gallon shall be computed by dividing total grams of lead used at a refinery in the manufacture of gasoline by total gallons of gasoline manufactured at such refinery.

(3) For each 3-month period (January through March, April through June, July through September, October through December) commencing with the period January 1, 1975 through March 31, 1975, each refiner shall submit to the Administrator a report showing for each refinery (i) the total grams of lead in lead additive inventory on the first day of the period, (ii) the total grams of lead received during the period, (iii) the total grams of lead in lead additive inventory on the last day of the period, (iv) the total gallons of gasoline produced by such refinery during the period, and (v) the average lead content in each gallon of gasoline produced during the period. Reports shall be submitted within 15 days after the close of the reporting period, on forms supplied by the Administrator upon request.

(b) The provisions of paragraph (a)(1) (i) and (ii) of this section shall not be applicable to any refiner which does not have more than 30,000 barrels per day crude oil or bona fide feed stock capacity from owned or leased facilities or from facilities made available to such refiner under an arrangement such as, but not limited to, an exchange agreement (except one on a refined product for refined product basis), or a throughput or other form of processing agreement, with the same effects as though such facilities had been leased.

4. A new § 80.25 is added as follows:

§ 80.25 Controls applicable to lead additive manufacturers.

For each 3-month period (January through March, April through June, July through September, October through December) commencing with the period January 1, 1975 through March 31, 1975, each lead additive manufacturer shall submit to the Administrator a report showing the total grams of lead shipped to each refinery by such lead additive manufacturer during the period. Reports shall be submitted within 15 days after the close of the reporting period, on forms supplied by the Administrator upon request.

5. A new § 80.26 is added as follows:

§ 80.26 Confidentiality of information.

Information obtained by the Administrator or his representatives pursuant to this part shall be treated, in so far as its confidentiality is concerned, in accordance with the provisions of 40 CFR Part 2.

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A 116

EPA'S POSITION ON THE HEALTH IMPLICATIONS OF AIRBORNE LEAD

PREPARED BY

U.S. ENVIRONMENTAL PROTECTION AGENCY
401 M STREET, S.W.
WASHINGTON, D.C. 20460

NOVEM 28, 1973

Appendix B

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I. Introduction

On February 23, 1972, EPA published a notice in the Federal Register (1), setting forth proposed regulations promulgating Federal standards for the use of lead and phosphorus additives in gasoline. Pursuant to the notice, several public hearings were held. In addition, numerous written comments were received by the Agency during an extended public comment period. After consideration of the hearing testimonies and written comments, and after further consideration of the available information on health effects of airborne lead, including the adverse effects of leaded gasoline on emission control devices, the regulations were divided into two separate pieces of regulatory action: final regulations providing for the general availability of lead-free gasoline were promulgated on January 10, 1973 (2) and regulations based upon the health effects of airborne lead, providing for the reduction of lead in leaded gasoline were reproposed (3). The regulations on reduction of lead in leaded gasoline for health reasons were reproposed because the Agency's basis for this reduction was substantially revised and had not been available for review by the scientific community.

At the time of the reproposal, the Agency was aware that there had not been unanimity either within or outside the

government with respect to the need to reduce lead in leaded gasoline. In light of this controversy, the Agency requested that informed and concerned members of the public provide EPA with their opinions on the matter. This commentary was encouraged by announcement in the Federal Register (3) and through approximately 150 individual letters specifically requesting comments from members of the scientific and industrial communities and from public interest groups.

As a result of these requests, numerous comments were received by the Agency. Although a substantive response to each comment was not possible, all comments were considered and are available for public inspection at the Freedom of Information Center of the Environmental Protection Agency in Washington, D.C.

The present paper is not intended as an encyclopedic treatment of all that is known about lead. Rather, it is intended to be a presentation of the pertinent evidence upon which a decision could be made as to whether or not there is a health justification to regulate lead in gasoline. While the emphasis in the paper is on lead as a gasoline additive, it has been necessary to consider other sources of lead in the environment in order to understand the role of leaded fuel in relation to human lead exposure. The Agency has also considered whether replacements for lead additives will result in deleterious health effects to as great or greater degree than the

lead additives themselves. This issue is discussed in a separate paper entitled, "Lead in Gasoline, Impact of Removal on Current and Future Automotive Emissions" (4).

The present paper was prepared by a committee of EPA scientists.

Members of the Committee Include:

John Buckley, Ph.D., Chairman

Kenneth Bridbord, MD
Douglas I. Hammer, MD
Robert Horton, MD
Marty Kanarek, MPH
Wellington Moore, DVM
Magnus Piscator, MD
Lawrence Plumlee, MD
Steven Reznek, Ph.D.
Richard Rhoden, Ph.D.
Jerry Stara, DVM

At least two committee members read and considered each of the approximately 130 comments received on the reproposed regulation. The results of this review process are reflected in the present paper as is additional information which has become available since reproposal of the regulation.

On October 1-2, 1973, EPA co-sponsored with the National Institute of Environmental Health Sciences a Conference on Low Level Lead Toxicity. A detailed evaluation of all presentations at the Conference is beyond the scope of the present document.

The proceedings from this Conference are due to be published in the near future.

The present document is an extension of the earlier paper entitled, "EPA's Position of the Health Effects of Airborne Lead," dated November 29, 1972. At the time the earlier paper was prepared, it seemed likely that catalytic converters would be the principal means of reducing pollutants from automobiles for a decade or more, and that the associated requirement of lead-free gasoline to protect the catalysts would result in near-elimination of automotive-related lead in the environment. It now appears that the impact of catalytic converters may be more transitory and that, unless regulated otherwise, lead additives will continue to be used in substantial amounts. Thus the health implications of leaded fuels become central to the regulatory decision process.

REFERENCES

1. Federal Register, Vol. 37, No 36; pp. 3882-3884, February 23, 1972.

2. Federal Register, Vol. 38, No. 6; pp. 1254-1256, January 10, 1973.

3. Federal Register, Vol. 38, No. 6; pp. 1258-1261, January 10, 1973.

4. Moran, J.B., "Lead in Gasoline, Impact of Removal on Current Automotive Emissions." Environmental Protection Agency, National Environmental Research Center, Research Triangle Park, North Carolina, October 1973.

II. Occurrence and Sources of Lead in the Environment

Lead occurs naturally in the earth's crust and is also found in the atmosphere and hydrosphere due to airborne dust, and to gases diffusing from the earth's crust (1). Lead was one of the first metals to be used by man, and evidence of the adverse health effects caused by lead and its compounds is found in the earliest annals of man's history. Deleterious effects from the present use of lead can be a problem, particularly in certain segments of the population. This Section will examine the influence of man upon the distribution of lead in the environment, especially with respect to the use of lead as a gasoline additive.

Lead or its compounds may enter and contaminate the environment at any step during the mining, smelting, processing, and use of the metal and its derivatives. The annual increase in lead consumption in the U.S. during the 10-year period 1962-1971 averaged 2.9%, resulting largely from an increased demand for batteries and gasoline additives (2). During the same period, lead used in pigments and caulking and in metal products declined. In 1971 the total lead consumption was 1,431,514 short tons with 596,797 short tons (42%) coming from recycled lead (3).

Because lead and many lead salts or oxides are relatively inert or immobile in the environment, a judgment can be made as to the ease with which lead from various use categories can reach and be transported through the environment. Table II-1 summarizes lead consumption patterns in the United States for 1968 and 1971 (4,5) according to categories of environmental impact. At this time, not all information required to compile a detailed lead balance for the United States is available; thus the above inventory of lead products in terms of their potential release to the environment must be recognized as subjective.

As shown in Table II-1, 352,614 short tons or approximately 25% of the lead consumed in 1971 was in the form of metallic lead or lead alloy. Most of this lead is chemically inert under typical environmental conditions. As long as the size of the piece of metallic lead or lead alloy is large, as is usually the case within the metal products category, it is relatively environmentally immobile. Recognized exceptions to this generalization do exist and include small pellets of lead shot often eaten by waterfowl, and the incineration of lead foils and tubes, with resultant release of lead into the atmosphere.

Storage batteries accounted for 679,803 short tons of lead consumption in 1971. This represents approximately 50% of the total. Since a large fraction of the lead in storage batteries

is recycled, little lead is believed to reach the environment from disposal of storage batteries.

Lead is also commonly used as a coating material. In 1971, some 53,198 short tons were in this category. Because of the possibility of erosion or wear, the lead thus used must be considered as potentially releasable into the environment.

More difficulty exists in classifying lead pigments in terms of their potential for reaching the environment. Lead pigments accounted for 81,258 short tons of lead consumption in 1971. Much of this amount is tabulated simply as red or white lead or litharge, rather than according to the actual category of use. Based on shipping information, approximately one-fourth of the lead classified under pigments in Table II-1 is used in the ceramics industry (6) and would thus have a relatively small environmental impact. However, this form of lead could contribute directly to human exposure through, for example, improperly glazed dinnerware.

Lead pigment is used in printing inks and paints. Lead in printer's ink, especially colored ink, has definite potential to reach the environment as a result of incineration. The use of lead-based paints in residential applications has been greatly reduced in recent years, although the total used in the past is still significant in terms of eventual environmental impact. The

amount of lead released into the environment from pigments is dependent on the relative quantity of waste disposal by incineration compared to other disposal methods, as well as wear and erosion of painted surfaces. On a national basis, approximately 10-20% of all solid waste is incincerated (7). This figure is assumed to be applicable as a first approximation to estimate the release of lead to the environment from incineration of lead pigments. The estimate does not necessarily account for all lead incineration from demolished buildings, since old paint tends to have a higher lead content. Further, this does not reflect entrance of lead into the environment from wear and erosion of exterior painted surfaces which has been shown to be substantial immediately adjacent to such surfaces. (8)

In 1971, 264,240 short tons of lead consumed were in the form of lead additives in gasoline. This amount is about one fifth of the total consumption for that year. It can be calculated that about 70% of the lead additives used in gasoline are emitted to the environment as particulate matter from the tailpipe (9). Much of the balance remaining within the engine or exhaust system of the vehicle is believed to eventually be removed from engine surfaces by transfer to the oil or by flaking of deposits in the exhaust system. The combustion of leaded gasoline accounts for by far the largest portion of all lead now reaching the environment, comprising about 90% of airborne lead emissions (9).

As evidence of this contribution, a 43% reduction in lead additives consumed within New York City during 1972 resulted in a 37% decrease in annual average ambient lead concentrations in the city for 1972 compared to 1970-1971 (10). In a more detailed calculation annual averages of air lead data from 38 sampling sites across the city were as follows: July 1969-June 1970, 1.62 $\mu g/m^3$; July 1970-June 1971, 1.71 $\mu g/m^3$; and July 1972-June 1973, 1.18 $\mu g/m^3$. The latter figure reflects the decrease in air lead following limitation of lead content of gasoline sold within the city to 1.0 gram per gallon on January 1, 1972. Though meteorologic differences from year to year, as well as sampling and analytic variability could have accounted for some of this decrease, the relatively constant city wide average in the two years preceding reduction combined with the decline following reduction make it evident that the contribution of lead additives to airborne lead in New York City is substantial.

Table II-2 (9, 11, 12) summarizes the major discharges of lead into the environment. Since current knowledge regarding the mobility or activity of lead in the environment is incomplete, the information in this table can only be regarded as semi-quantitative. Of the total amount of lead found in the atmosphere, the contribution from natural sources, such as the re-entrainment of surface soil in the form of dust, is small. This contribution has been estimated to be about 0.0005 $\mu g/m^3$ of air.(13) The lead concentrations in today's urban atmospheres are

at least 100 to 1000 times greater than the amount which can be attributed to natural sources.

As noted above, over two thirds of the lead added to gasoline reaches the air environment (14). As lead leaves the exhaust pipe of an automobile, initially all of it is airborne. The heaviest particles fall to the ground within several hundred feet of roadways,(15,16), whereas others remain suspended for longer periods of time. The length of time that lead particles emitted in auto exhaust remain airborne is determined primarily by particle size and weight as well as by meteorological factors. Lead is removed from the air by gravitational settling of larger particles, aggregation and subsequent settling of smaller particles, and through scavenging by various forms of precipitation.

Fallout of automobile exhaust lead contributes to lead levels found in dust and dirt which may in turn be resuspended in air. Surveys of street and sidewalk dirt in Washington, D. C. and in Boston showed lead concentrations commonly in the range of 1000 to 2500µg/g (ppm) with some values as high as 5000-10,000µg/g (17, 18). Samples of sidewalk dirt taken in the Bedford-Stuyvesant area of Brooklyn, New York over a five month period ranged from 900 to 4900 ppm lead (19).

In a study of 77 midwestern cities, the average concentrations of lead in dust collected at residential, commercial, and industrial sites were 1,636 µg/g, 2413 µg/g, and 1512 µg/g respectively (20). Lead concentrations in surface soil from three California city parks ranged from 194 to 3,357 µg/g (21).

Lead levels in urban housedust commonly ranged from 1000-2000 µg/g in Boston (22) and averaged 500-900 ppm in Brattleboro, Vermont, being higher in homes near roadways (23). In middle class residential areas of New York City, lead in house dust averaged 600-700 µg/g (24) or 2-3 times higher than similar measurements made in a suburban community. Lead emissions from automobiles, and industrial sources, as well as weathering of leaded paint all contribute to this contamination.

A very small part of the atmospheric lead resulting from lead fuel additives is in an organic form (gaseous lead alkyls). Alkyl lead concentrations are rarely as high as 10% of ambient air lead values (25, 26). Higher but transient peaks of alkyl lead concentration have been demonstrated near specific sources such as spills of gasoline containing lead alkyl compounds.

Air lead concentrations have been shown to be closely related to the density of vehicular traffic, being highest in large cities and progressively lower in the suburbs, smaller towns and

rural areas (27). Data from non-urban sampling sites in the National Air Surveillance Network (NASN) showed mean air lead concentrations of approximately 0.1-0.5$\mu g/m^3$ during 1953-1966. The mean concentrations at urban sites for those years were 1-3$\mu g/m^3$.

In Pasadena and Los Angeles, air lead concentrations sampled for two to seven hours have averaged from 9.4 to 23.6 $\mu g/m^3$ during weekday traffic and have ranged up to 29.3 $\mu g/m^3$ during the morning rush hour (28). Studies at different locations of the Queens Midtown Tunnel and Triborough Bridge in New York City showed that ambient lead levels averaged from 2.1 to 35.6 $\mu g/m^3$ per 24 hours with individual 24 hour peaks ranging up to 57.3 $\mu g/m^3$ (29). About 68% to 88% of these airborne lead particulates were in the respirable range.

One study has shown that air lead concentrations measured during 1968-69 in Los Angeles, Philadelphia, and Cincinnati increased 56%, 19%, and 17%, respectively, over concentrations measured in 1961-62 (30). The average lead concentration consistently increased at 17 of the 19 sampling sites and meteorological conditions at each sampling site were consistent for the two time periods. NASN data, which comes largely from the central city commercial areas, does not show such a marked increase during the same period, perhaps because traffic

densities were consistently high in these areas in the 1961-1969 period.

The extent to which airborne lead has affected the lead content of the earth's waters is difficult to ascertain. Lead enters the aquatic systems through precipitation, lead dust fallout and erosion and leaching of soil, as well as through municipal and industrial waste discharges. In addition, deposited lead (fallout) is washed off of streets, highways, and other surfaces into rivers, lakes and marine waters. Extrapolations from recent studies (31,32) indicate that on a national basis the contribution of lead from fallout washing off streets into water as urban runoff may be as high as 5,000 tons each year. Today, surface waters in the Mediterranean and Pacific Oceans contain up to 0.20 and 0.35 µg of lead/kg water respectively, which is about 10 times greater than the estimated pre-industrial lead content of marine water (33). The lead content of rivers and lakes also has increased in recent times. (33) A study of rainfall in various cities of the NASN network indicated an average concentration of 36 µg of lead/liter of rain which was approximately 40 times as high as that found in rainwater at non-urban sites (34).

Of concern is the possibility that continued contamination of the environment by atmospheric lead has affected the

concentrations of lead in biological systems and subsequently in man's diet. Data suggest that some biological systems are subjected to increased concentrations of lead in their environment and certain portions of the food chain (earthworms) have shown increases in lead content. (35). Shellfish that are eaten by man are known to take up and store lead. Vertebrate animals tend to store absorbed lead in bone rather than in flesh, and therefore do not so readily transmit lead up the food chain.

The quantitative contribution of atmospheric lead to the food chain of man is not known. Lead is a natural, although minor, constituent of plants. Plants can absorb lead to varying degrees (36). The solubility of lead is important when considering lead uptake by plants. Experimental studies have shown that plants can absorb and translocate only the soluble forms of lead (37). There have been no reported observations that would indicate that plants in the field are adversely affected by atmospheric lead. The most important aspect of lead fallout on plants is the potential hazard to humans or animals from the consumption of contaminated and unwashed foliage, fruit or roots. In areas with heavy lead dustfalls, such as along busy highways, substantial amounts of lead are found on exposed surfaces of plants. Since much of the atmospheric lead particulate is insoluble, a large portion of the lead contamination remains on the surfaces of plants. There is evidence of this source being important in lead poisoning of grazing animals (38). To the extent that lead

remains when plants are consumed by humans, it can add to man's total body burden of lead.

Marine species, with the exception of shellfish, usually do not concentrate lead from seawater and, hence, would not contribute much to human lead exposure. In one study, eastern oysters, soft-shelled clams, and northern quahogs were shown to concentrate lead from seawater (39). Although concentrations of lead reported in shellfish (range 0.17-2.5 ppm) are not high, they do indicate the ability of certain edible aquatic species to concentrate lead from the surrounding medium (40). Lead levels in soft clams, hard clams, and surf clams have at times been found that exceeded the maximum acceptable level proposed by federal experts (41).

The World Health Organization reports that according to the results of total diet studies in industrialized countries, the total intake of lead from food generally ranges from 200-300 µg per person per day. WHO further states that based upon available data, these levels are similar to those found in the past 30-40 years and that no upward trend in lead levels in food is evident (42).

Though lead in food would be the most consistent contributor to total lead exposure for the general population, lead in food is not the source that is most reduceable in the event that total

exposure to lead is found to be undesirable. For the general population, "the lead in air is probably the contribution that is most accessible to action for reducing the total body burden of lead especially where this fraction is large compared with that absorbed from food." (43) Lead levels in food, of course, still require careful monitoring to assure that excess exposure does not occur from this source.

In conclusion, man made sources of lead contribute most to the lead which enters the environment. Of these sources, the combustion of leaded gasoline is the single most significant contributor. Potentially, human exposure to environmental lead may result from a number of sources, among them being the inhalation of airborne lead and the ingestion of lead-contaminated dusts. Reducing the amount of airborne lead in the environment constitutes an accessible means for reducing potential human exposure to environmental lead.

TABLE II-1 (4,5)

LEAD CONSUMPTION (SHORT TONS)
(UNITED STATES)

Product		1968	1971
Metal Products			
Ammunition		82,193	87,567
Bearing metals		18,441	16,285
Brass & bronze		21,021	20,044
Cable covering		53,456	52,920
Casting metals		8,693	7,281
Collapsible tubes		9,310	10,041
Foil		6,114	4,417
Pipes, traps & bends		21,098	18,174
Sheet lead		28,271	27,607
Type metal		27,981	20,812
Weights & ballast		16,768	17,453
Solder		74,074	70,013
	TOTAL	367,420	352,614
Storage Batteries			
Grids & posts		250,129	322,236
Oxides		263,574	357,567
	TOTAL	513,703	679,803
Coatings & Miscellaneous			
Caulking lead		49,718	29,993
Annealing		4,194	4,068
Galvanizing		1,755	1,395
Plating		389	582
Terne metal		1,427	1,409
Other		17,924	15,751
	TOTAL	75,407	53,198
Pigments			
White lead		5,857	4,731
Red lead & litharge		86,480	61,838
Pigment colors		14,163	13,916
ZnO		3,234	773
	TOTAL	109,734	81,258
Chemicals			
Gasoline additives		261,895	264,240
Misc. chemicals		629	401
	TOTAL	263,155	264,461
GRAND TOTAL		1,329,419	1,431,514

TABLE II-2 (9, 11, 12)

LEAD EMISSIONS (TONS) TO THE ENVIRONMENT FOR 1968
(UNITED STATES)

Atmospheric		
Gasoline combustion		181,000
Coal combustion		920
Lead alkyl manufacturing		810
Secondary smelting		811
Incineration of solid wastes		11,000
Other		755
	TOTAL	195,316 1/
Water		
Printing inks		10
Petroleum storage		24
Paint production		320
Battery production		600
	TOTAL	954
Insecticides		2,081
	GRAND TOTAL	198,351

1/ It is estimated that 5,400 tons of lead due largely to automotive lead emissions deposited on city streets reach the aquatic environment. Additional, but unknown, amounts are washed out of the atmosphere by precipitation and land directly on rivers, lakes and marine water.

REFERENCES

1. Airborne Lead in Perspective, A Report Prepared by the Committee on Biological Effects of Atmospheric Pollutants, National Research Council, National Academy of Sciences, Washington, D. C., 1972, p.5.

2. Ryan, J.P., Minerals Yearbook 1971. U.S. Department of the Interior. U.S. Government Printing Office, Washington, D.C., p. 671.

3. Ibid., p. 684.

4. Ibid., p. 684.

5. Airborne Lead in Perspective, op.cit.; p. 11.

6. Ryan, J.P., op.cit., p. 686.

7. Klee, A.J., et.al., "Preliminary Data Analysis, 1968, National Survey of Community Solid Waste Practices." USPHS Publication #1867, Solid Waste Program, Cincinnati, Ohio.

8. Comments submitted by the Ethyl Corporation to the Environmental Protection Agency, March 9, 1973, pp. V-16 - V-19.

9. Airborne Lead in Perspective, op.cit., pp. 11-13.

10. Personal communication from Fred C. Hart, Commissioner of Air Resources, New York City, to William D. Ruckelshaus, Administrator of EPA, March 20, 1973 and personal communication from Edward F. Ferrand, NYC Department of Air Resources to Kenneth Bridbord, EPA, Oct. 19, 1973.

11. "Recommended Methods of Reduction, Neutralization Recovery or Disposal of Hazardous Waste," Prepared by TRW Systems Group, EPA, Report #21485-6013-RU-00, February 1, 1973, Vol. I, p. 92-94.

12. The Pesticide Review, 1972, U.S. Department of Agriculture, Agriculture Stabilization and Conservation Service, Washington, D. C.

13. Airborne Lead in Perspective, op.cit., p. 5.

14. Ibid., pp. 11-13.

15. Motto, H.L., R.H. Daines, D.M. Child, and C.K. Motto, Lead in Soils and Plants. Its Relationship to Traffic Volume and Proximity to Highways. Environ. Sci. Tech. 4:231-238, 1970.

16. Airborne Lead in Perspective, op.cit., p. 29.

17. Krueger, H.W., Krueger Enterprises, Cambridge, Mass. Testimony submitted to EPA, July 10, 1972.

18. Fritsch, A., and M. Prival, Center for Science in the Public Interest, Washington, D. C., Testimony submitted to EPA, 1972.

19. Lombardo, L.V., The Public Interest Campaign, Washington, D. C., Personal Communication to Mr. William D. Ruckelshaus, Administrator, U.S. Environmental Protection Agency, March 9, 1973.

20. Airborne Lead in Perspective, op.cit., p. 139.

21. Ibid, p. 30.

22. Krueger, H.W., op.cit.

23. Darrow, D.K. and Schroeder, H.A., "Childhood Exposure to Environmental Lead," paper presented before the American Chemical Society, Chicago, Ill., August 29, 1973.

24. Pinkerton, C., J.P. Creason, D.I. Hammer and A.V. Colucci, "Multi-media Indices of Environmental Trace Metal Exposure in Humans," presented at 2nd Int. Symp. on Trace Element Metabolism in Animals, Madison, Wisconsin, June 1973.

25. Airborne Lead in Perspective, op.cit., p. 193.

26. U.S. Department of Health, Education and Welfare, Public Health Service, Division of Air Pollution. Survey of Lead in the Atmopshere of Three Urban Communities. PHS Pub. No. 999-AP-12. Cincinnati: Public Health Service, 1965. pp. 57-58.

27. Airborne Lead in Perspective, op.cit., pp. 18-21.

28. Ibid, p. 19.

29. Ferrand, E.F., Assistant Commissioner for Scientific Affairs, New York City Department of Air Resources, Testimony submitted to EPA April 11, 1972.

30. Tepper, L.B. and Levin, L.S. A Survey of Air and Population Lead Levels in Selected American Communities. Department of Environmental Health, College of Medicine, University of Cincinnati, Cincinnati, Ohio. Final Report (Contract PH-22-68-28), December 1972. 73 pp.

31. Water Pollution Aspects of Street Surface Contaminants, Environmental Protection Technology Series, EPA-R2-72-081, November 1972, p. 3.

32. Environmental Pollution by Lead and Other Metals, NSF Rann Grant 6I-31605, University of Illinois at Urbana-Champaign Progress Report May 1 - October 31, 1972, Chapter 6.

33. Airborne Lead in Perspective, op.cit., p. 6.

34. McCabe, L.J., J.M. Symons, R.D. Lee, and G.R. Robeck, Survey of Community Water Supply Systems. J. Am. Water Works Assoc. 62(11): 670-687, 1970.

35. Gish, C.D. and R.B. Christensen, "Cadmium, Nickel, Lead and Zinc in Earthworms from Roadside Soil", Environmental Science and Technology, 7:1060-1062, November 1973.

36. Airborne Lead in Perspective, op.cit., p. 33-37.

37. Ibid., pp. 33-37.

38. Ibid., pp. 178-182.

39. Pringle, B.H., D.E. Hissong, E.L. Katz, and S. Mulawka, Trace Metal Accumulation by Estuarine Mollusks. J. Sanit. Eng. Div.; Proc. Amer. Soc. Civil Eng. 94:455-475, 1968.

40. Airborne Lead in Perspective, op.cit., p. 189.

41. Metals in Shellfish with Particular Reference to Lead. Prepared by the Northeast Water Supply Research Laboratory of the U.S. Environmental Protection Agency, May 19, 1972, p. 17.

42. Joint FAO/WHO Expert Committee on Food Additives, Evaluation of Certain Food Additives and the Contaminants Mercury, Lead, and Cadmium; WHO Technical Report Series, No. 505, 1972, p. 19.

43. Ibid., p. 20.

III HEALTH ASPECTS OF LEAD EXPOSURE

It is apparent from our knowledge of the distribution of lead in the geosphere and biosphere that lead in small amounts is a natural part of living things. Lead enters animals from plants which pick it up from the soil. In spite of its ubiquity, no functional requirement for lead has been clearly demonstrated in living organisms.

Because lead is easy to extract and purify, it has been used for centuries. Lead, if improperly employed, is capable of producing serious poisoning and even death. All of the situations in which it is harmful are of human origin. For example, no known cases of lead poisoning have originated from natural amplification in the food chain. Much of our knowledge of lead poisoning comes from occupational exposures to the metal or its compounds and from ingestion of lead by children.

The commonest symptoms of lead poisoning are anemia, severe intestinal cramps, paralysis of nerves, particularly of the arms and legs, and fatigue. The symptoms usually develop rather slowly. High levels of exposure produce severe neurologic damage, often manifested by encephalopathy and convulsions. Such cases are frequently fatal. Today, severe cases of lead poisoning in occupational situations are rarely seen due to improved working conditions. Lead encephalopathy is now predominantly found in young children who have consumed flakes of

peeling lead paint, and also in some people who consume illegal whiskey, which often contains lead.

Exposure to lead is usually assessed by means of lead levels in blood and urine. Ordinarily, blood lead levels in the general population are between 10 and 30 µg/ 100g of whole blood. The expressions "µg/100g of blood" and µg/ 100ml of blood" are for all practical purposes equivalent and will be used interchangeably in this paper, depending upon which is used in the original references. Clinical symptoms of lead poisoning usually do not appear until blood lead levels reach 80-100 ug/100g or higher. Symptomatic lead poisoning may, however, be associated with blood lead levels in the range of 50-60 µg/100g in instances where the patient's response to lead in the tissues is modified as for example in the presence of anemia (1).

When the most severe effects of lead poisoning, e.g.encephalopathy, have occurred and not been fatal, recovery is frequently incomplete (2). Permanent, irreversible damage to the nervous system often results, even though no further high level exposure occurs. With less severe effects such as anemia and intestinal symptoms, residual, irreversible injury is not observed if the exposure is discontinued. Recovery may be slow, however.

Most injurious substances produce less conspicuous effects at exposure levels somewhat below those resulting in overt symptoms.

These less conspicuous effects must therefore be specifically searched for by examinations or tests; otherwise, they may not be observed. Lead is strongly suspected to produce such subtle effects, but they have been much less well documented than have the more conspicuous ones.

The suggestion that subtle lead effects occur comes in part from studies of overt lead toxicity in which residual neurologic effects were found during followup examinations among children who initially were not deserved to have such symptoms. It should be noted, however, that the problem of subclinical lead effects is distinct from that of residual neurologic damage following lead poisoning. Whether subclinical lead effects themselves can result in residual neurologic damage is another question which has not been adequately resolved.

In this regard, a study involving 425 children with lead poisoning attributed primarily to ingestion of lead-based paint found that 39% showed evidence of nervous system damage during follow-up examinations (3). Mental retardation and recurrent seizures were the most common and persistent findings. In many instances neurologic symptoms were not found at initial examinations. This study, however, dates back to 1955, when diagnosis of lead poisoning often depended upon tests for urinary coproporphyrins, which are not usually positive below 80-100 µg of lead/100 g. whole blood. It is therefore probable that large amounts of lead had been ingested. Extrapolation of these

results tc children with blood lead levels below 80 µg/100g is not appropriate.

Smith, et al (4) examined the neurologic sequelae in children diagnosed with lead poisoning at least five years prior to study. Permanent neurologic damage was found only in children who had lead poisoning accompanied by encephalopathy. Subtle damage due to lead exposure, however, could not have been recognized in this study since sophisticated psychological and fine motor function tests were not employed.

More recent studies have employed a greater degree of sophistication in testing behavioral changes associated with exposure to lead. Kotok et al (5) studied children with similar socio-economic backgrounds to determine whether increased lead body burden interferes with normal neurologic and motor development. A group having a mean blood lead level of 81 µg/100 g was not found to differ significantly from a control group having a mean blood lead of 38 µg/100 g. On this basis, it was concluded that blood lead levels below those usually associated with encephalopathy do not interfere with childhood development. The mean blood lead level of the control group selected, however indicated significant exposure to lead. Thus, firm conclusions cannot be drawn from these data regarding whether or not lead adversely affects the nervous system at blood lead levels of 80µg/100g or below.

Albert, et al. (33) employed psychological tests, medical examinations, nerve conduction studies and school records to evaluate whether deleterious effects could be associated with childhood lead exposure below overt toxicity. Excess lead exposure was measured in terms of lead levels in teeth. In the absence of a diagnosis of lead poisoning or a blood lead level in excess of 60 µg/100g, high levels of lead in teeth were not associated with deleterious health effects. In constrast, adverse effects were seen in children with blood lead levels in excess of 60 µg/100g who received no chelation therapy as well as among these who were treated for severe lead poisoning.

A study by Pueschel et.al. (6) suggests that in children lead body burdens below those usually associated with clinical poisoning contribute to renal as well as neurological damage. Fifty-eight children between the ages of 6 months and 6 years were selected for study from a larger sample of children living in a high-risk lead poisoning area. The majority of these children were known to ingest paint and dirt. The children examined had blood leads over 50 µg/100ml or had blood leads over 40 µg/100ml with urine leads exceeding 500 µg/24 hours following EDTA provocation.

While most of the children did not exhibit symptoms uniquely attributable to lead poisoning, over one-third were found to have a history of mild CNS symptoms. The symptoms were observed significantly more frequently in these children than in a matched control group. Subsequent follow-up study revealed that many of

the children examined still had behavioral and school difficulties, even though sources of lead exposure had been removed in most cases. Since complete data on the control groups were not presented, it is difficult to fully evaluate the significance of these findings.

Of particular interest in Pueschel's work is the observation of anemia, aminoaciduria, proteinauria, and casts in a high percentage of the children. It may be noted that studies by Betts, et. al. suggest anemia can occur in children with blood lead levels between 37-60 µg/100ml and studies by Tola, et al. found mild anemia occurring in lead workers with blood leads between 40-60 µg/100g (7-8). Although factors such as nutritional status and deprived environments may have been responsible for the anemia and renal and neurological damage observed by Pueschel, these effects are consistent with damage that has been related to lead exposure.

It has been suggested by David, et al. (9) that childhood behavioral disturbances such as hyperactivity may be associated with blood lead levels between 25-55 µg/100ml. About half of the hyperactive group studied (28 of 54) had blood leads between 25-55 µg/100ml compared to less than one third (10 of 37) in the control group. Both the hyperactive and the control groups were similar with respect to age and sex, but socio-economic, racial and dietary differences between the groups may have confounded the results. Further, the children studied for hyperactivity may have had much higher lead absorption at an earlier age with a

subsequent decline in blood lead level by the time of study. Because the hyperactive child is more likely to have pica (abnormal ingestion of non-food substances), it is possible that the hyperactivity caused the increased lead intake rather than having been the consequence of it. The David study suggests, although it does not prove, that exposure to lead in quantities not generally considered excessive or toxic may contribute to hyperactivity in children. Additional studies are necessary to define the relationship between hyperactivity and causal factors, including lead. Studies by Silbergeld, et al. (34) may be important in this regard. Mice exposed to high levels of lead from birth developed hyperactivity which, as in children with this disorder, responded atypically to CNS stimulants and depressants.

A study by de la Burde, et al. (10) concluded that children ingesting paint or plaster containing lead are more likely to have abnormal or suspect behavior and fine motor disabilities than children not so exposed. Subjects in this investigation were followed prospectively and were selected to eliminate pre-existing neurologic abnormalities. The lead-exposed group had a mean blood lead level of 58 µg/100 g (range 40 to 100 µg/100g); blood leads were not measured in the controls. Lead intake differences between the groups were established on the basis of measures of urinary coproporphyrins.

Results indicate that the control group performed better than the exposed group in the areas tested. Most notable differences

were with respect to fine motor ability and behavior. These results, however, may have been confounded by the failure to measure blood leads in the controls and by the failure to exclude, in the exposed group relative to the controls, an increased incidence of pica. Increased behavioral deficiencies observed in the exposed group could have been related to a greater frequency of pica in this group rather than the ingestion of lead per se.

Some studies have recently been undertaken to determine the lowest exposure levels at which lead-induced injury to the peripheral nerves can be detected (11-13). One study shows that in adults disturbances in conduction velocity can be found at blood lead levels of 80 µg/100g and above. Preliminary results also noted in this study indicate the presence of mild neuropathy in lead workers at blood lead levels under 50 µg/100 g (13).

The potential problem of subclinical neurological damage caused by lead is an important one which deserves further attention. Whether subtle adverse effects on CNS function such as small decreases in IQ, interference with fine motor function and altered behavior can be attributed to lead exposure is still uncertain. If such effects do occur they may be related to a degree of exposure less than that recognized as producing overt lead poisoning.

In recent years evidence has been accumulated that significant exposure to lead will produce injury involving the

kidneys and other organ systems. This knowledge of complex and varied effects has recently been reviewed by the National Academy of Sciences (NAS). Table III-1 which is reproduced here from Table 4-11 of the NAS review of lead displays a detailed outline of effects at various exposure levels (14). It should be noted that some of these effects are more typically associated with lead exposure in children than in adults.

Because anemia is an evidence of lead toxicity, blood and blood cell formation have been studied particularly for early changes; shortened red cell life span (15) and lowered hemoglobin (16) have been observed. Injury to enzyme systems involved with the formation of hemoglobin has also been observed, but, because some of these systems are disturbed by substances and conditions other than lead, the observations are difficult to interpret. One biochemical change, indicated by an increased urinary excretion of delta aminolevulinic acid (ALA-U), is of particular interest because it is rarely produced by anything other than lead (17). This increase in ALA-U is one of the most thoroughly studied of the metabolic alterations produced by lead. It is directly related to inhibition of the enzyme delta-aminolevulinic acid dehydrase (ALAD) which converts ALA to porphobilinogen. Elevated ALA-U has been shown to be closely related to elevated lead levels in the soft tissues and may reflect biochemical changes in these tissues (18-19). Soft tissue lead is considered to be the metabolically active portion of the body lead burden.

(20) For these reasons, increased ALA-U is considered to be an undesirable change indicative of probable health risk (21).

The appearance of excess ALA in the urine is first seen at blood lead levels of 40-60 µg/100g (22-25). If increased urinary ALA excretion is undesirable, it then follows that blood lead levels above 40 µg/100g are to be avoided, if possible. In this context, the National Academy of Sciences Lead Panel concluded (26), "...the exponential increase in ALA excretion associated with blood lead content above approximately 40 µg/100g of blood signifies inhibition of ALAD that is physiologically signif cant in vivo."

Recent animal experiments, using graded levels of lead exposure, show beginning injury to kidneys at about the same exposure level as that causing increased ALA-U excretion (27). While these kidney changes are probably reversible when mild, it is known that permanent damage to the kidneys results from higher doses of lead (28). The degree and duration of exposure needed to produce irreversible changes are not known, although it is unlikely that they could be caused solely by lead in the ambient air.

Animal studies indicate that lead at much higher dosages than man would generally be exposed to is capable of disturbing the biological defense mechanisms that provide protection from bacterial and viral infection (29-31). The mechanism by which lead aggravates viral disease is by reduced interferon synthesis

rather than by inhibition of interferon action (32). Because it has become apparent that lead may produce this type of injury, further studies, with expected exposure routes and realistic exposure levels, should be of considerable interest.

The studies reviewed in this Section permit no unequivocal conclusions to be drawn. On balance, the studies suggest that subclinical changes may be associated with blood lead levels in the range of 40-60 µg/100g. As blood lead levels increase above 40 µg/100g, the likelihood that significant changes will occur increases markedly. Based upon evidence from these studies, it would seem prudent to regard blood lead levels over 40 µg/100g as indicators of lead intake that should be prevented. The 40 µg/100g figure, however, does not represent a sharp demarcation between health and disease.

TABLE 4-11 Level and Types of Effects of Inorganic Lead Salts as Related to Estimates of Various Levels of Absorption–Recent and Remote

Type of Effects	Level I: No Demonstrable *in vivo* Effect	Level II: Minimal Subclinical Metabolic Effect	Level III: Compensatory Biologic Mechanisms Invoked	Level IV: Acute Lead Poisoning		Level V: Late Effects of Chronic or Recurrent Acute Lead Poisoning
				Mild	Severe	
Metabolic (accumulation and excretion of heme precursors)	Changing ALAD[a]	Slight increase in urinary ALA may be present	ALA, UCP, FEP progressively increased	ALA, UCP, FEP increased 5- to 100 fold		Increased if excessive exposure recent, but may not be increased if excessive exposure remote
Functional injury: Hematopoiesis	None	None known	Shortened red-cell life-span, reticulocytosis (±) (reversible)	Shortened red-cell life-span and reticulocytosis with or without anemia (reversible)		Anemia (±) (reversible)
Kidney (renal tubular function)	None	None known	?	Amino-aciduria, glycosuria (±) (reversible)	Fanconi syndrome (reversible)	Chronic nephropathy[b] (permanent)
Central nervous system	None	None known	?	Mild injury (??? reversible)	Severe injury (permanent)	Severe injury[b] (permanent)
Peripheral nerves	None	None known	?	Rare	Rare	Impaired conduction (wrist, foot drop usually improve slowly, but may be permanent)
Clinical effects	None	None known	Nonspecific mild symptoms (may be due in part to coexisting diseases)	Colic, irritability, vomiting	Ataxia, stupor, coma, convulsions	Mental deficiency (may be profound), seizure disorder, renal insufficiency (gout) (permanent)
Index of level of recent or current absorption:						
Blood lead, μg/100 g of whole blood	<40	40–60	50–100+	>80 With anemia, intercurrent disease: 50–100+	>80	May be normal
Urine lead (adults only), μg/liter	–	<80	<130	>130 (May be less in severe illness)	>130	Spontaneous excretion may be normal

[a]See p. 106 for discussion of changing levels of ALAD.
[b]CaEDTA mobilization test in chronic nephropathy is positive; may or may not be positive in permanent central nervous system injury.

REFERENCES

1. "Airborne Lead in Perspective," Report prepared by the Committee on Biologic Effects of Atmospheric Pollutants, National Research Council, National Academy of Sciences, Washington, D.C., 1972, p. 130

2. "Airborne Lead in Perspective," op. cit., p. 95

3. Perlstein, M.A., and R. Attala, "Neurologic Sequelae of Plumbism in Children Clin. Ped. 5:292-298, 1966

4. Smith, H.D., Baehner, R.A., Carney, T. and Majors, W.J., "The Sequelae of Pica With and Without Lead Poisoning." Amer. Jour. Dis Child. 105: 609, 1963

5. Kotok, D., Development of Children with elevated blood lead levels, Prediat. 80: pp. 57-61, Jan 1972

6. Pueschel, S.M., Kopito, L, and Sohwachman, H., Children with an increased lead burden, Jour. Amer. Med. Assoc. 222:4:462-466, Oct. 23, 1972

7. Betts, P.R., Ashley R. and Raine, D.N., "Lead Intoxication in Children in Birmingham,"Brit. Med. Jour., 1:pp. 402-406, 1973

8. Tola, S., Hernberg, S., Asp, S., and Nikkanen, J., "Parameters Indicative of Absorption and Biological Effect in New Lead Exposure: A Prospective Study," Brt. J. of Ind. Med., 30: pp. 134-141

9. David, O., Clark, J. and Vohller, K., "Lead and hyperactivity" Lancet, 2: 900-903, October 28, 1972

10. de la Burde, B., and M.S. Choate, Does asymptomatic lead exposure in children have latent sequelae? Pediat. 81:6:1088-1091, December 1972.

11. Catton, M.J., G. Harrison, P.M. Fullerton and G. Hazantzis, Subclinical neuropathy in lead workers, Brit. Med. Journ. 2: 80-82, 1970.

12. Seppalainen, A.M. and S. Hernberg, Sensitive technique for detecting subclinical lead neuropathy, Brit. Journ. Indust. Med. 29: 443-449, 1972.

13. Hernberg, S., Biological effects of low lead doses, Proc. Int. Symp. Envir. Hlth. Aspects Lead, CED, CID, pp. 617-629, Luxembourg, May 1973.

14. Airborne Lead in Perspective, op. cit., pp. 128-129.

15. Hernberg, S, M. Nurminen and J. Hasan, Nonrandom shortening of red cell survival times in men exposed to lead, Environ. Res. 1:247-261, 1967.

16. Williams, M.K., Blood lead and hemoglobin in lead absorption, Brit. Jcurn. Indust. Med. 23:105-111, April 1966.

17. Airborne Lead in Perspective, op. cit., p. 106.

18. Cramer, K. and S. Selander, Studies in lead poisoning: Comparison of different laboratory tests, Brit. Jour. Indust. Med. 22: 311-314, 1965.

19. Selander, S., K. Cramer and L. Hallberg, Studies in lead poisoning: oral therapy with penicillamine. Relationship between lead in blood and other laboratory tests, Brit. Journ. Indust. Med. 23: 282-291, 1966.

20. Airborne Lead in Perspective, op. cit., p. 123.

21. Airborne Lead in Perspective, op. cit., p. 132.

22. Selander S. and K. Cramer, Interrelationships between lead in blood, lead in urine, and ALA in urine during lead work, Brit. Jour. Indust. Med. 27: 28-39, 1970.

23. Hernberg, S, J. Nikkanen, G. Mellin and H. Lilius, Delta-aminolevulinic acid dehydrase as a measure of lead exposure, Arch. Envir. Hlth. 21:140-145, 1970.

24. Tola, S., The effect of blood lead concentration, age, sex, and time of exposure upon erythrocyte deltaaminolevulinic acid dehydrase activity, Work Envir. Hlth. 10: 26-35, 1973.

25. Tola, S., S. Hernberg, S. Asp and J. Nikkanen, "Parameters indicative of absorption and biological effect in new lead exposure: a prospective study," Brit. J. Ind. Med., 30: pp. 134-141, 1973.

26. Airborne Lead in Perspective, op. cit., p. 110.

27. Goyer, R.A., J.F. Moore and M.R. Krigman, Lead dosage and the role of the intranuclear inclusion body, Arch. Envir. Hlth. 20: 705-711, 1970.

28. Airborne Lead in Perspective, op. cit., p. 131.

29. Selye, H., B. Tuchweber and L. Bertok, Effect of lead acetate on the susceptibility of rats to bacterial endotoxins, Journ. Bacteriol., 91: 884-890, 1966.

30. Hemphill, F.E., M.A. Kaeberle and W.B. Buck, Lead suppression of mouse resistance to Salmonella typhimurium, Science 127: 1031-1032, June 4, 1971.

31. Gainer, J.H., "Effects of Metals on Viral Infections in Mice," Env. Health Persp., pp. 98-99, June 1973.

32. Gainer, J.H., "Lead Aggravates Experimental Viral Infections and Represses the Antiviral Activity of Interferon Inducers," paper presented at EPA-NIEHS Conference on Low Level Lead Toxicity, Raleigh, N.C., Oct. 1-2, 1973.

33. Albert, R.E., R.E. Shore, A.J. Sayers, C. Strehlow, T.J. Knelp, B.S. Pasternak, A.J. Friedhoff and J. Goodgold, "Followup of Children Overexposed to Lead," paper presented at EPA-NIEHS Conference on Low Level Lead Toxicity, Raleigh, N.C., Oct. 1-2, 1973.

34. Silbergeld, E.K. and A.M. Goldberg, "Hyperactivity: A Lead-Induced Behavior Disorder," paper presented at EPA-NIEHS Conference on Low Level Lead Toxicity, Raleigh, N.C., Oct. 1-2, 1973.

IV. Can an Acceptable Lead Body Burden be Defined?

In Section III the spectrum of effects due to lead was reviewed with special emphasis upon subclinical changes believed to be of health significance. This Section will examine how these findings may be of assistance in helping to define an acceptable lead body burden for the general population.

Exposure to toxic substances is difficult to quantify with external measurements, especially when exposure may occur via food, water and air. It is preferable, if possible, to measure an integrated index of these exposures within the body. The blood level of lead which itself is not an adverse health effect serves as such an index, and, under most circumstances is a reasonably accurate measure widely employed in public health surveillance. This measurement serves not only as an index of continuous lead exposure, but also as a diagnostic tool in identifying cases of lead poisoning and undue lead absorption.

Due to the ubiquitous nature of lead, the general population carries blood lead levels of 10-30µg/100g. As exposure increases, blood lead levels rise, although slowly and in a nonlinear fashion, because excretion of lead also rises with increasing exposure. Thus blood lead levels are a satisfactory index of soft tissue lead burden during constant exposures, but

are less accurate during rapid changes in lead exposure or in total body burden.

Blood lead concentration is the net result of several independent equilibria. Accordingly a single blood lead determination will not necessarily indicate a high nor exclude a low body burden of lead. Serial determinations are therefore recommended to assure the validity of the initial value as well as to ascertain rates of change (1). The variability of laboratory methodology and accuracy is also often considerable, and the confidence limits of the analytical procedure must be taken into account especially when comparing results among different laboratories.

Although physicians are generally well aware of limitations with the use of blood lead levels, the even greater variability of other indices and the ease of obtaining blood specimens make determination of blood lead the single most useful parameter. Other indices are helpful, including: concentration of lead in urine, feces, and hair; ALA in the urine; urinary coproporphyrin; erythrocyte delta aminolevulinic acid dehydrase, protoporphyrin, basophilic stippling, and red cell survival time; hemoglobin; and hematocrit. A detailed discussion of the relative merits of these techniques is beyond the scope of the present paper.

As noted in Section III, blood lead levels of 40µg/100g and above have been associated with subclinical changes as well as increased lead body burdens, and as such are considered to be evidence that significant lead intake has occurred. This 40 µg/100g level has been accepted as evidence of undue exposure to lead in children and adults (2,3,4), although one must recognize that this level does not represent a sharp demarcation between health and disease.

The U.S. Public Health Service has recommended (3) that, for older children and adults, until proven otherwise by additional research, "... a blood lead concentration of 40µg or more per 100ml of whole blood, ... determined on two separate occasions, be considered evidence suggestive of undue absorption of lead, either past or present." This recommendation also established 80µg/100ml whole blood as unequivocal lead poisoning and 50-79µg/100ml as requiring immediate evaluation as a potential poisoning case.

The FEP test (free erythrocyte protoporphyrins test) developed by Piomelli et al (11), shows promise of identifying the presence of undue metabolic effects resulting from lead exposure. The FEP test is capable of detecting an interference in heme synthesis caused by lead in the bone marrow. This interference, if present to a significant degree, could reflect a degree of lead exposure associated with toxic metabolic effects.

The FEP test is especially valuable since it is well recognized that blood lead levels do not, in themselves, constitute an absolute criterion of lead effect.

There exists debate within the scientific community with respect to what concentration of FEP in the blood constitutes an abnormal elevation. As originally proposed by Piomelli, an FEP level of 250 µg/100ml of RBC appeared a reasonable upper limit above which significant adverse effects due to lead were likely occurring. This 250 µg/100ml cutoff is approximately three times the FEP level found in 1-6 year old children with blood leads under 20 µg/100ml. There are, however, scientists who advocate a lower FEP cutoff to provide a greater margin of safety in any screening program.

Piomelli found that all children studied with blood leads of 60 ug/100ml or above had FEP levels in excess of 250 µg/100 ml of RBC. Among children with blood lead levels between 40-59 µg/100ml, 55% were observed to have FEP levels above 250 µg/100ml. In contract, only 5% of children with blood leads below 40 ug/100ml had elevated FEP levels. In this regard, one must consider the possibility that iron deficiency anemia potentiates lead toxicity even at blood lead levels below 40 ug/100g (12). The widespread prevalence of iron deficiency anemia among the population at greatest risk from lead exposure should also be noted. A positive FEP test accompanied by a blood

lead below 40 µg/100ml urually reflects the presence of iron deficiency anemia although this could also be an early indicator of undue lead absorption. The finding that 55% of children with blood leads between 40-59 µg/100ml have elevated FEP levels, combined with the exponential increase in FEP observed at blood leads above 40 µg/100ml, suggests the more frequent occurrence of adverse metabolic effects among children whose blood leads have risen to 40 µg/100ml and above. Combined with blood lead determinations, the FEP test would provide a more objective evaluation of lead toxicity than possible by either test alone. It may well be that subclinical neurological effects due to lead are more closely related to FEP levels in the hematopoietic system than to isolated measures of blood lead (13).

Environmental insults may put certain population subgroups such as children, the aged or infirm, and pregnant women at greatest risk. Although there is no clear evidence of increased susceptibility to lead in children, they may be considered a potentially high risk group because they have greater opportunity to ingest lead from environmental sources such as paint, dust or dirt. High lead exposure gives rise to renal tubular damage in children, which is an unusual consequence in adults. In children, excessive exposure is frequently associated with sudden onset of encephalopathy which is often followed by permanent brain damage (5). The onset of encephalopathy without previous symptoms is uncommon in adults.

Angle and McIntire (6) concluded that there is a definite fetal risk, maximal in the first trimester, from intrauterine exposure to high concentrations of lead in maternal blood. Studies by Kochen and Haas et al (7,8) confirm that umbilical blood lead concentrations are similar to those in maternal blood. In the newborn the blood-brain barrier is relatively immature (9,10) and the mass of central nervous system tissue is greater, while the mass of skeletal tissue is less, per unit of body weight. With the well documented neurotoxicity of lead, it is clear that additional data are necessary to evaluate the possibility of increased susceptibility to lead exposure in the fetus and neonate.

Two recent experiments are suggestive of a special susceptibility to lead among animals exposed either prenatally or during infancy. In one instance (14), lambs exposed to maternal blood lead levels of 34 µg/100ml during gestation demonstrated slowed learning on a visual discrimination task at 10-15 months of age. This deficit is consistent with visual-perceptual problems that have been noted in children with lead poisoning. Extrapolation of these findings directly to man is, however, somewhat uncertain since a blood lead of 34 µg/100ml in sheep may be equivalent to a higher blood lead in man. In another instance (15), infant rhesus monkeys fed 0.5 mg lead acetate per kg of body weight developed hyperactivity and insomnia. Blood leads in these infant monkeys ranged from 60-100 µg/100ml. In contrast

the physical status and behavioral status was unchanged in juvenile monkeys fed lead acetate at 20 mg/kg sufficient to produce average blood leads of 135 µg/100ml. Though lead dosages administered to the monkeys were well in excess of those found in human lead poisoning, the dependence of response upon age is noteworthy.

It is not possible at this time to firmly establish a single acceptable blood lead level protective of all high risk population subgroups. It would appear prudent, however, to recommend that the current U.S. Public Health Service guideline for older children and adults, i.e., 40µg/100ml whole blood, be regarded as a strict upper limit for younger children. Whether the acceptable upper limit should be lower than 40µg/100ml for the fetus, neonate, and the woman of child bearing age will require further investigation.

Important questions that necessitate additional study to help define with greater confidence an acceptable lead body burden include the following:

(1) At what level of exposure or body burden do effects of lead become irreversible?

(2) Are some of the permanent effects not immediately recognizable, especially in children?

(3) When the less severe effects subside is there no residual, or are there some permanent changes which are not noticeable?

(4) What combinations of level and duration of exposure prcduce irreversible lead poisoning?

(5) Does injury to other organs or systems such as the liver or biological defense mechanisms occur at levels of exposure and lead body burden common among the general population?

(6) At what level of exposure and lead body burden are changes due to lead found which produce no symptoms and will such changes if long continued produce irreversible damage?

(7) Which groups among the general population are most sensitive to lead and which factors can significantly affect lead toxicity?

Considerably more research is thus necessary to clarify these as well as other incompletely answered questions related to lead.

REFERENCES

1. Lin Fu, J.S., "Undue Absorption of Lead among Children - A New Look at an Old Problem," N. Eng. J. Med., 286 (13), 702-710, 1972.

2. "Airborne Lead in Perspective," report prepared by the Committee on Biological Effects of Atmospheric Pollutants, Division of Medical Sciences, National Research Council, National Academy of Sciences, Washington, D.C. 1972, pp. 100-110

3. "Medical Aspects of Childhood Lead Poisoning," HSMHA Health Reports, 86(2), 140-143, 1971

4. Chisolm, J.J., Baltimore City Hospitals, in testimony submitted to EPA, July 26, 1972.

5. Lin Fu, J.S., op cit.

6. Angle, C.R., and M.S. McIntire, "Lead Poisoning During Pregnancy," Amer. J. Dis. Child., 103, 436-439, 1964.

7. Kochen, J.A., Mentifiore Hospital and Medical Center,

personal communication to K. Bridbord, EPA, April 18, 1973.

8. Haas, T.; K. Mache; K. Schaller; A. Wieck; W. Mache; and H. Valentin; "Untersuchungen Uber Die Okologishe Bleibelastung Im Kindesalter;" Proceedings of the International Symposium on the Environmental Health Aspects Lead, CEC, CID, Luxembourg, May, 1973, pp. 741-748.

9. Dobbing, J.: The development of the blood brain barrier Prog. Br. Res. 29: 417-27, 1968

10. Evans, C.A. et al.: The development of blood brain barrier and choroid plexus function in immature foetal sheep, J. Physiol 224:15-16, July 1972

11. Piomelli, S., B. Davidow, V. F. Guinee, P. Young and G. Gay, The FEP (Free Erythrocyte Protoporphyrins) Test: A screening Micromethod for Lead Poisoning, Pediatrics, 51:254-259. February 1973, and Letters to the Editor, Pediatrics 51:303-306. March 1973.

12. Angle, C. R., and M. S. McIntire, "Red Cell Lead, Whole Blood Lead and Red Cell Enzymes," Paper presented at EPA-NIEHS Conference on Low Level Lead Toxicity, Raleigh, North Carolina, October 1-2, 1973.

13. Chisolm, M M., D. Mellits, J. E. Keil and M. B. Barrett, Variations in Hematologic Responses to Increased Lead Absorption in Young Children, paper presented at EPA-NIEHS Conference on Low Level Lead Toxicity, Raleigh, North Carolina, October 1-2, 1973.

14. Carson, T. L., G. A. Van Gelder, G. G. Karas and W. B. Buck, Development of Behavioral Tests for the Assessment of Neurologic Effects of Lead in Sheep," paper presented at EPA-NIEHS Conference on Low Level Lead Toxicity, Raleigh, North Carolina, October 1-2, 1973.

15. Allen, J. R., P. J. McWey and S. J. Suomi, "Pathobiological

and Behavioral Effects of Lead Intoxication in the Infant Rhesus Monkey," paper presented at EPA-NIEHS Conference on Low Level Lead Toxicity, Raleigh, North Carolina, October 1-2, 1973.

V. Relationship of Environmental Lead Exposure to Human Lead Intake

In Section II it was demonstrated that lead emitted from motor vehicles reaches the environment in substantial quanities by several routes including lead suspended in air and fallout of lead into dust and onto soils. This widespread dispersal of lead into the environment resulting from the presence of lead in gasoline, may contribute to man's intake of lead. The routes of intake of lead into man from the environment are through ingested food and non-food items, water, dust, and air inhalation. This present Section examines the extent to which man's lead intake could be affected by lead emissions from motor vehicles and what effect, if any, this may have upon the people so exposed.

Lead is added to gasoline as tetraethyl lead, an organic compound. Exposure to organic lead is dangerous and an important occupational health problem. As indicated in Section II, a very small part of the atmospheric lead resulting from fuel additives is in an organic form (gaseous lead alkyls) and there is little opportunity for general population exposure. General population exposure is much more likely to come from inorganic lead contained in the aerosol particulate emitted from automobiles.

To more fully understand the factors affecting man's lead intake and any resulting biomedical effects, it is useful to discuss the routes of lead absorption, the quantity of lead available through each route, and the differential absorption among the various routes. It is in this context that the sources of lead intake can be examined with respect to the highest amount of total daily lead intake that is generally considered safe or acceptable over a long period of time. For the general population, absorption of lead through the skin is nil. The lead that is swallowed into the gastrointestinal tract is available for absorption regardless of source, but only a small portion is actually absorbed. Similarly, lead inhaled into the respiratory tract is available for absorption through the lungs or from the gastrointestinal tract after it has been moved to the throat by ciliary action and subsequently swallowed. Only a portion of the inhaled lead is absorbed.

Commonly accepted figures for daily dietary lead intake in adults are on the order of 200-300 µg/day with ranges of 100-500 µg/day (1, 2). Gastrointestinal absorption of lead is primarily a function of the chemical composition of the diet and is usually estimated to be 10%, although it has been reported to be lower in adults (3, 4). Young children, however, may absorb considerably more lead, up to 50% of their oral lead intake (5). Recent experiments in animals indicate that diets low in calcium greatly increase lead absorption from the gastrointestinal tract

and affect lead distribution in the body with more lead being stored in the soft tissues (6). Diets low in calcium and low in iron have both been shown to increase lead toxicity in rats fed subtoxic quantities of lead in drinking water (7).

Respiratory absorption of lead is dependent upon the size, shape, and density of the inhaled particles as well as the rate and volume of respiration. For respiratory absorption, 30% is a commonly accepted figure, assuming essentially complete pulmonary retention of deposited lead particles of the size found in ambient air. (8) Other estimates of respiratory lead absorption have ranged from 17% to 37% (9, 10). Thus, for equal amounts of available lead, an average of about three times as much of a given amount of lead would be absorbed from the respiratory tract as from the gastrointestinal tract. Table V-1 summarizes the results of EPA's calculations to estimate the contribution of airborne lead to total daily lead absorption among adults in the general population. A lead isotope tracer technique developed by Rabinowitz et al. has been helpful in further clarifying the relative contribution made by airborne lead to total lead absorption in man (11). Preliminary results from these isotope studies are consistent with the calculations in Table V-1. Since, as noted in Section II, lead in gasoline is by far the largest contributor to airborne lead, the calculations in Table V-1 indicate that lead in gasoline can make a substantial contribution to lead absorption in man.

In the classic experiments which he summarized in the 1960 Harben lectures, Kehoe fed known amounts of lead to four subjects and monitored their daily intake and output of lead as well as the lead concentrations in their blood and urine. (12) Each subject had a daily diet containing about 300 µg of lead to which additional lead was added in the following quantities: approximately 300 µg (Subject S. W.), 1000 µg (Subject M. R.), 2000 µg (Subject E. B.), and 3000 µg (Subject I. F.). The average daily total intake for each study subject was 600 µg, 1270 µg, 2350 µg, and 3270 µg respectively. Gastrointestinal absorption was determined to be about 10% of the dietary lead dose. In his Harben lectures Kehoe plotted the average blood lead level for each subject for consecutive 28-day periods during the course of the study. Although Kehoe did not give the regression lines of blood lead versus time for each subject, they can be calculated from the data in his original figure, reproduced as Figure V-1 (12). The source data from Subject S. W. for the calculation of this line are retabulated in Table V-2.

The slope of the regression line for Subject S. W. while ingesting a total of 600 µg of lead each day was significantly different from zero ($t=2.82$, p less than .01), whereas the slope was not significantly different from zero during his control period. The actual regression line as calculated from the data in Table V-2 and shown in Figure V-2 is as follows:

$$y = 36.5 + 0.70 (x - 7.5)$$

where y = predicted whole blood lead level at time x

and

x = time in 28-day periods

The best fit for these data may be a curvilinear rather than a linear function, in which case, blood leads would level off about 40 µg/100g rather than continuing to increase indefinitely.

This equation predicts that a blood lead concentration of 40 µg/100g would be reached in 12.5 28-day time periods with a 95% confidence interval ranging from 8.9 to 16.1 time periods. In other words, a subject ingesting only a 300ug lead supplement to the 300 ug in his diet would theoretically reach a blood lead level of 40 µg/100g in just less than one year. This prediction may, of course, be dependent upon the relatively high blood lead baseline (30-32 µg/100g) in subject S.W. when supplemental lead feedings were initiated and may not accurately reflect any additional lead intake in subject S.W. due to airborne lead. It should also be noted that this 600ug figure is consistent with the World Health Organization's maximum recommended intake of lead for adults of 50µg of lead/kg/week, the equivalent of 500µg of lead intake daily for a 70kg man (13).

As described in Section III, urinary delta-aminolevulinic acid excretion begins to increase as blood lead content rises above a level of about 40µg/100g. Such an increase in urinary ALA excretion is generally considered indicative of biochemical changes in the tissues that are undesirable and physiologically

significant (14). Based upon Kehoe's data, a daily lead ingestion of 600µg (with an absorption of 60µg) could cause blood lead to increase to a level associated with increased urinary ALA excretion (i.e., 40µg of lead/100g). In this context, one can estimate the additional daily lead intake in the diet or in the air necessary to increase lead in blood to this level.

Calculations of this nature are presented in Table V-3. If average dietary intake is estimated to be 200-300 µg of lead, or an absorbed amount of 20-30 µg daily, an ordinary man would have to regularly absorb only an additional 30-40 µg of lead daily to reach a biologically undesirable blood lead level. Assuming 10% absorption, he could do this by an additional dietary ingestion of 300-400 µg daily, or by combinations of dietary ingestion and inhalation. The main concern here, however, is what air lead exposure level could also result in an increased absorption of 30-40 µg daily?

The daily amount of lead absorbed from inhaled air is the product of: the airborne lead concentration; the volume of air inhaled daily; and the percent of lead reaching the respiratory tract that is absorbed. The airborne lead concentration varies from place to place. The volume of air inhaled daily has been estimated at about 23 cubic meters (m^3) for a "standard man" (weighing 70kg, 20--30 years of age, 175 cm tall, and having a surface area of 1.8 m^2) engaged in light work; about 85% of this

air being inhaled during waking hours (15). Other estimates have been lower, in the range 15-20m^3 of inhaled air daily. As an example, the amount of lead absorbed from breathing air containing 1$\mu g/m^3$ of lead assuming 30% absorption and inhalation of 20m^3 of air each day, is 6.0μg. For a constant dietary lead intake, exposure to increasing airborne lead levels will increase both the absolute amount of lead absorbed by this route and the relative proportion of total lead absorption into the blood which comes from air.

Air levels necessary to increase a man's total daily lead absorption by 30 and by 40μg are estimated in Table V-4. A range of values was calculated by using different parameters for daily air intake and daily lead absorption from the respiratory tract. These calculations indicate that a daily air lead exposure of 5.0$\mu g/m^3$ for a standard man could increase his daily lead absorption by 30μg. Such Ambient air lead levels attributable largely to lead in gasoline have been noted in portions of several U.S. cities at the present time (16).

Experimental data on humans exposed to particulate lead in a chamber support these theoretical calculations (17, 18). The chemical composition and size of the lead oxide particles in these experiments were not identical to those emitted from automobiles; nevertheless, these differences were sufficiently

small so that inferences can be made regarding effects of airborne lead among the general population.

Volunteer subjects in New York State living in a chamber were exposed to airborne lead levels of 10.9µg/m^3 and 3.2µg/m^3 for 23 hours a day in two separate experiments. Average blood lead levels among the men exposed to 10.9µg/m^3 of lead daily increased from about 20 to 37µg/100ml of lead in whole blood after 125 days (18 weeks); average blood lead levels among a second group of men exposed to 3.2µg/m^3 of air lead for 11 weeks increased from about 19 to 25µg/100 ml of lead in whole blood.

These results are compared to those of Kehoe (12) referred to earlier in Table V-5. Estimates of the daily total lead absorption for the study subjects breathing lead at 10.9µg/m^3 and Dr. Kehoe's subject S.W. are shown in this table. The results of both experiments are similar and, while the New York State subjects might have had a somewhat greater total daily lead absorption, their average blood lead levels increased from 20-37µg/100ml in only a little over four months.

The experiment in New York State at lead exposure levels of 3.2µg/m^3 also showed that the men who had been previously exposed to airborne lead at 10.9µg/m^3, responded differently to their subsequent re-exposure, compared to subjects not so previously exposed. (18) The previously exposed men had consistently higher

blood lead levels before and after exposure to 3.2$\mu g/m^3$ than the men without such exposure. This observation is consistent with Goyer's recent suggestion that repeated exposure to lead may alter pathways of lead detoxification and excretion (19).

Several epidemiologic studies have investigated the relationship between blood lead levels and airborne lead exposures. A study, commonly referred to as "The Seven City Lead Study", showed a positive but not statistically significant correlation between average blood lead levels and average annual airborne lead exposures ranging in concentration from 0.17 to 3.39 $\mu g/m^3$. (20). Exposure was estimated from measurements made at sampling stations generally located within one mile of the homes of subjects being studied. Based upon consistent differences between average blood lead levels in urban and suburban dwellers, the authors concluded, "It is probable that these observations partially reflect lead absorption from ambient atmospheres differing in lead concentration. . . but that factors other than the atmospheric lead level are of relatively greater importance in determining the blood lead levels in population groups." This conclusion was strengthened by further analysis of the study results (21) which found that air lead was a significant though not the most influential factor affecting blood lead levels.

A study by Daines et al. obtained more accurate exposure information by studying the effect of distance of residence from

highways upon blood lead levels among black females (22). Average annual air lead levels on the front porches of homes located 3.7, 38.1, and 121.9 meters away from a highway were 4.60, 2.41, and 2.24 $\mu g/m^3$ respectively. There was no significant difference between the outside air lead concentrations at 38 and 122 meters, but both were different from the air lead concentration at 3.7 meters from the highway. This rapid non-linear decrease in airborne lead with distance from the source has been observed for mobile as well as stationary lead sources. Concentrations of lead in dustfall also decrease rapidly with increasing distance from the source. (23-25) Average annual air lead levels in the front room of houses at the three sampling points also reflected this non-linear decrease being 2.30, 1.50, and 1.57 $\mu g/m^3$ as the distances increased. Average blood lead levels in the study subjects were 23.1, 17.4, and 17.6 µg Pb/100 g at 3.7, 38.1, and 121.9 meters respectively. The average blood lead levels at the two more distant sampling sites differed significantly from the average at 3.7 meters, but did nct differ significantly from each other.

It is of interest to note that among the homes closest to the highway (3.7 meters), air lead and blood lead levels were significantly lower for subjects whose homes were air-conditioned than for similar subjects in homes without air-conditioning. Another finding was that significant differences in air lead, but not in blood lead levels were observed for subjects in homes at

33.4 and 457 meters from a turnpike (22,26). The average outdoor air lead levels at 33.4 meters and 457 meters were 1.95 and 1.73 $\mu g/m^3$; blood leads at these sites averaged 15.7 and 16.1 µg/100g respectively. In this experiment the air lead levels, though statistically different, would not necessarily be reflected in higher blood lead levels, as daily respiratory lead absorption nearer the turnpike would only amount to about 1 ug of lead per day more than at the distant site.

Azar et al. studied thirty male subjects in each of five locations in the United States (27). Air lead levels were measured with personal samplers twenty-four hours a day for two to four weeks and the combined air lead exposure each day ranged from 0.81 - 1.10 $\mu g/m^3$ for office workers in Starke, Florida, and Barksdale, Wisconsin, to 3.06 for office workers and 6.10 $\mu g/m^3$ for taxi drivers in Los Angeles, California. The average daily air lead exposure for a group of Philadelphia taxicab drivers was 2.62 $\mu g/m^3$.

Biweekly blood samples were obtained during the two taxicab studies and the Los Angeles office workers study; at Starke and Barksdale, a single blood specimen for each study subject was obtained at the beginning and at the end of the study. Limitations in the study design were summarized by the authors who wrote, "The use of different occupational groups located in different cities; studying each group at a different time; the

lack of data relative to ingested lead; and the lack of detailed histories and physical examinations are obvious design deficiencies in this study." Average blood lead levels ranged from 13.8 µg/100 g in Barksdale to 24.6 µg/100 g for the Los Angeles taxi drivers. Although the relationship was not linear, average blood lead values were generally positively associated with increasing airborne lead exposure. The study also found that 79-90% of the combined total airborne particulate lead exposure was in the respirable range, which is in agreement with the results from New York City. (28)

Jones et al. studied blood lead and carboxyhemoglobin levels among London taxi drivers on both the day and the night shift (29). Significant differences were found in blood carboxyhemoglobin, but not in blood lead levels, when comparing smoking and non-smoking day-shift drivers to similar night-shift drivers. The authors concluded that the day drivers had a greater exposure to motor car exhaust than the night shift drivers because they had significantly higher blood carboxyhemoglobin levels and therefore "it would seem that little of the lead found in their blood is attributable to the lead they inhale whilst driving in London streets." Unfortunately, both differences in smoking intensity between smoking groups, as well as actual differences in air and dietary lead exposure could explain these results and neither was measured. Furthermore, information was not obtained regarding the exposure of the

drivers when they were off-duty. Thus the authors' original conclusions failed to consider at least three pertinent confounding variables.

Two studies of blood lead levels among non-occupationally exposed people living in smelter communities provide additional data which support a relationship between blood lead levels and exposure to lead in air or dustfall. Average blood lead levels among 10 year old United States school boys living in smelter and non-smelter communities reflected environmental exposure rankings, but no quantitative measurements of lead in air or lead in dustfall were given.(30) However, the study pointed out that lead fallout on home grown produce did not explain the observed differences and concluded that increased lead exposure and absorption probably occurred by the respiratory as well as the gastrointestinal route. A study of adults living around a secondary lead smelter in Finland showed that blood lead levels were negatively correlated with the log of the distance from the source in meters (31). This study did publish isopleths of monthly dustfall lead in relation to the point source, but no air lead values were given. Nevertheless, both studies indicate a qualitative relationship between blood lead levels and exposure to lead in air and in dustfall.

Observed relationships between lead in air and lead in blood did not always fit a simple straight line in the above cited

epidemiologic studies. Using the previously stated assumptions for a "standard man", a person breathing 1 µg of lead/m^3 of air would absorb a total of 6 µg daily from his respiratory tract and a person breathing 3 µg of lead/m^3 of air would absorb a total of 18 µg daily from his respiratory tract. Ultimately this difference in absorption would be reflected in different blood lead levels providing other sources of lead intake were equivalent.

Assuming 10% lead absorption from the gastrointestinal tract and a range of normal daily dietary lead intake from 100-500 µg, total lead absorption from the gastrointestinal tract would range from 10-50 µg each day. The need to measure dietary lead intake in studies of air lead effects is important since one would not be able to distinguish a person (Subject A) breathing 1 µg/m^3 of lead in air and eating 340 µg of lead daily from a person (Subject B) breathing 3 µg/m^3 of lead in air and eating 220 µg of lead daily, viz:

Lead Intake	Subject A	Subject B
Absorbed from air	6 µg	18 µg
Absorbed from diet	34 µg	22 µg
Total Daily Lead Absorption	40 µg	40 µg

Furthermore, variations among individuals with respect to gastrointestinal as well as respiratory lead absorption must be considered in evaluating such data.

Since none of the epidemiologic studies of air lead and blood lead considered both lead in diet and variability of gastrointestinal and respiratory absorption among individuals when evaluating air lead-blood lead relationships, it is understandable why simple straight line relationships between the two variables are not consistently found. As exposure to airborne lead increases, dietary variations would become less likely to confound true differences in blood lead due to air lead exposure.

The above calculations were made for a "standard man;" comparable calculations for women are difficult since respiration and absorption data for women are less complete than those for

men. While the total dietary intake and total daily respiration of women are generally less than those of men, women generally are smaller with proportionately lower caloric and oxygen requirements. Thus the relative relationships with respect to lead absorption from the gastrointestinal and respiratory tracts should be rather similar to those for men. It is even more difficult to extrapolate such calculations to children because they are in a phase of rapid growth and the parameters of interest change in a non-linear fashion. For example, a one year old child has only about 1/7 the body weight of an adult, yet has about 1/3 to 1/4 the total daily air intake ($6m^3$) and about 40-60% of an adult's total dietary lead intake (130 µg) (32). Hence, a child takes in less lead on a total basis, but proportionately more lead on a body weight basis than an adult. At present, the ultimate effect of these differences upon lead intake and absorption in children when compared to adults is not fully known.

Lead from automotive exhaust which is deposited in city dust and dirt also is a potential source of exposure for human beings. A gram of dust or dirt would be only about 1/4 teaspoon, although this may vary somewhat depending on its characteristics. Since these dusts often contain 1000-2000 µg/g of lead or more, the safe intake level of lead for children would be greatly exceeded if only a small fraction of a teaspoon of dust were swallowed daily. (33)

In summary, lead from automotive exhaust contributes to human exposure both from the air and from fallout. Blood lead levels exceeding 40 µg/100 g are considered medically undesirable and may ultimately be harmful. Blood lead levels depend on daily dietary intake and absorption of respired lead. For a "standard man" with average dietary lead intake, exposure to airborne lead concentrations of 5.0 to 6.7 $\mu g/m^3$ could cause his blood lead concentrations to reach 40 µg/100 g within a year. Airborne lead levels near to or in excess of 5 $\mu g/m^3$ have been observed in several U. S. cities.

Lead in dust and dirt poses another important source of exposure, especially for small children. Daily ingestion of relatively small quantities of dirt or dust (less than 1 gram or about 1/4 of a teaspoonful) containing 1,000-2,000ppm of lead would be medically undesirable. Although dustfall exposure to lead is still often considered an hypothesis, much of the available evidence is consistent with this hypothesis. Thus, present day lead exposure to air or dusts in some sections of large cities leaves little or no margin of safety compared to those concentrations associated with biomedical harm.

FIGURE V-1

The average concentration of lead in the blood of subjects S.W., M.R., E.B., and I.F. (from reference 12)

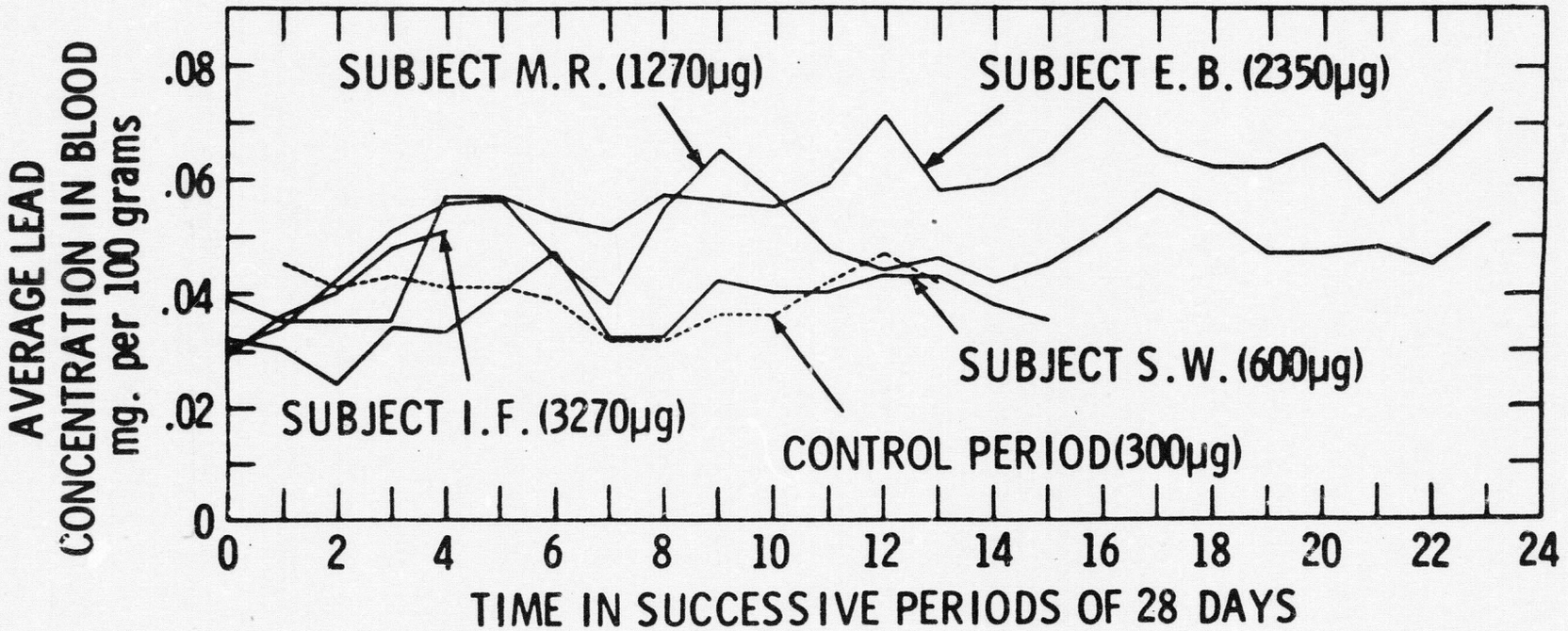


FIGURE V-2

Regression of average blood lead versus time in a subject ingesting a total of 600ug of dietary lead daily (adapted from reference 12)

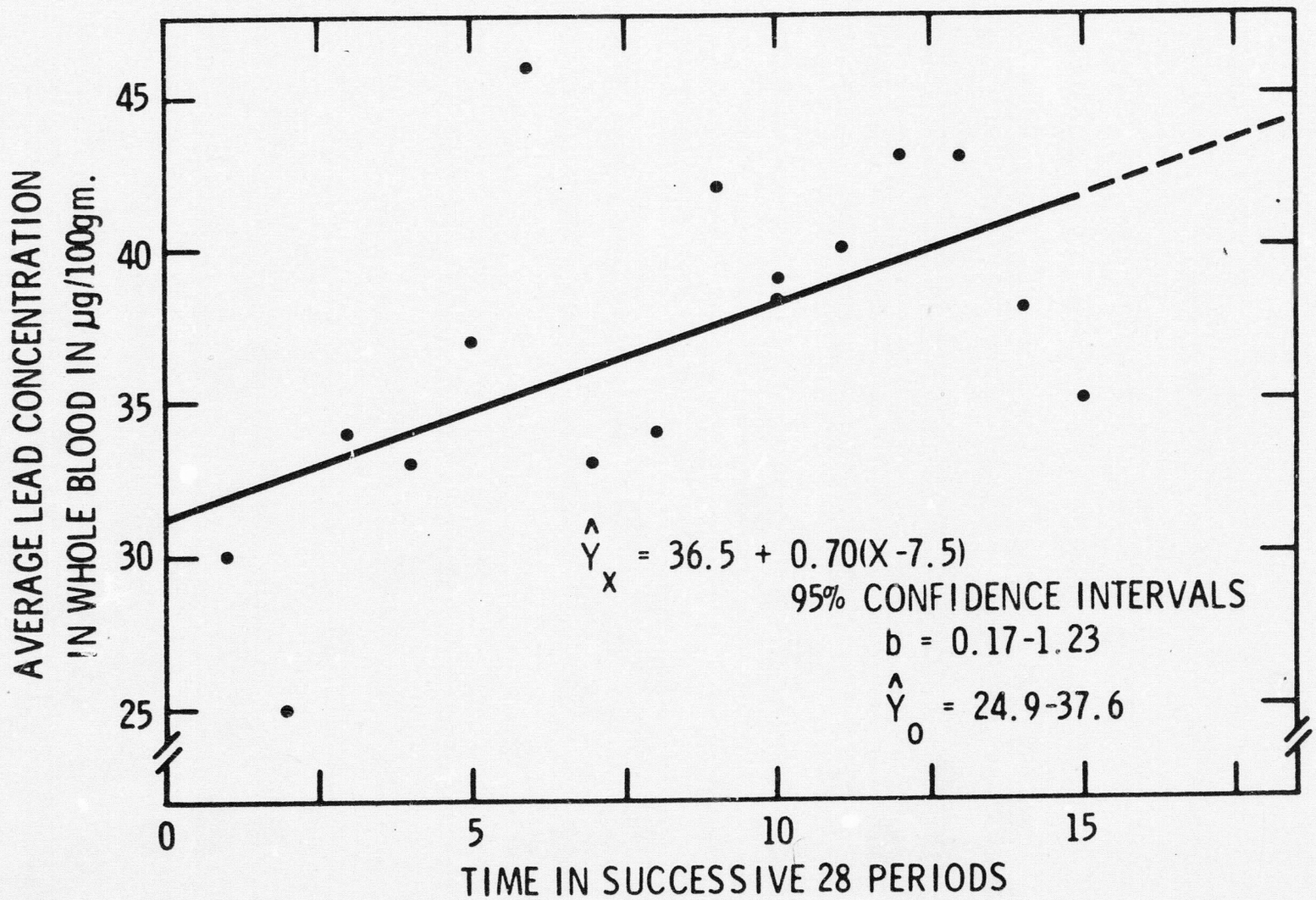


TABLE V-1

Amount of Lead Absorbed from Air
in Relation to
Airborne Lead Concentrations
Among Adults

Lead in Air ($\mu g/m^3$)	Daily Lead Absorbtion		Percent From Air
	From Air+	Total*	
0	0	30	0
1	6	36	17
2	12	42	29
3	18	48	38
4	24	54	44
5	30	60	50

+Assumes 30% absorption and 20m^3 daily respiration.

*Assumes 10% absorption of a daily dietary intake of 300 µg of lead.

TABLE V-2

Regression of Average Blood Lead Level
Versus Time in a Subject* Ingesting a Total of
600 µg of Dietary Lead Daily

x	y
0	32
1	30
2	25
3	34
4	33
5	37
6	46
7	33
8	34
9	42
10	39
11	40
12	43
13	43
14	38
15	35

where:

x = time in 28-day periods

y = average whole blood lead level for a 28-day period in µg/100 g of blood.

$y = 36.5 + 0.70 (x - 7.5)$

$S_x = 4.76$
$S_y = 5.53$
$S_{y.x} = 4.56$

*Reference 12. Subject S. W., average blood lead values taken from Figure 10., Lecture II.

TABLE V-3

Calculated Difference in Daily Lead Intake
Between a Biologically Undesirable Amount and the
Amount in the Normal Adult Diet

	Daily Lead Intake	
	Ingested	Absorbed
Biologically Undesirable Amount+	600 µg	60 µg
Normal Adult Diet	200-300 µg	20-30 µg
Difference	300-400 µg	30-40 µg

+Continued daily intake of this amount could cause blood lead levels to rise to about 40 µg/100 g of whole blood with the consequent increased excretion of delta-aminolevulinic acid in the urine.

TABLE V-4

Estimated Airborne Lead Exposures Which Could Cause
Adult Male Blood Lead Levels to Reach
40 µg/100 g of Whole Blood

Common Daily Dietary Lead Absorption	Increase Required to Reach 60 µg Daily+	Airborne Lead Exposures Which Would Cause the Designated Increase		
		Standard(a) Man	Least(b) Case	Highest(c) Case
20 µg	40 µg	6.7 µg/m^3	15.7 µg/m^3	5.4 µg/m^3
30 µg	30 µg	5.0 µg/m^3	11.8 µg/m^3	4.0 µg/m^3

+Experimentally in a human, absorption of 60 µg of lead daily caused his blood lead levels to reach 40 µg/100 g of whole blood. (12)

(a) Standard Man = 20m^3 x 30% absorption
(b) Least Case = 15m^3 x 17% absorption
(c) Highest Case = 20m^3 x 37% absorption

TABLE V-5

Estimated Daily Total Lead Absorption
in Two Experimental Studies

Exposure Source	Daily Lead Absorption New York (17,18) Chamber Exposure	Kehoe's Subject (12) S.W. Exposure
Diet	10 µg*	30 µg*
Experimentally Added	49-60 µg+ (Air)	30 µg* (Diet)
Daily Total Lead Absorbed	59-70 µg	60 µg

*Assumes 15m^3 daily respiration (since the men in both experiments were sedentary) and 30-37% absorption of 10.9 ug/m^3 of lead in air.

*Assumes 10% absorption of dietary intake.

REFERENCES

1. Joint FAO/WHO Expert Committee on Food Additives; "Evaluation of Certain Food Additives and the Contaminants Mercury, Lead and Cadmium; WHO Technical Report Series, No. 505, 1972.

2. Airborne Lead in Perspective, A Report Prepared by the Committee on Biologic Effects of Atmospheric Pollutants, National Academy of Sciences, Washington, D. C., 1972 p.50.

3. Airborne Lead in Perspective, op.cit., pp. 52-53.

4. Thompason, J.A.; Balance Between Intake and Output of Lead in Normal Individuals. Brit. J. Industr. Med., 28:189-194, 1971.

5. Alexander, F.W., H.T. Delves, and B.E. Clayton, the uptake and excretion by children of lead and other contaminants, Proceedings of An International Symposium on the Environmental Health Aspects of Lead, CEC, CID, Luxembourg, May 1973, pp. 319-331.

6. Goyer, R.A., and B.C. Rhyne, "Pathological Effects of Lead," Int'l. Rev. Exp. Path., 1973, 12:pp. 1-77.

7. Ibid.

8. Airborne Lead in Perspective, op.cit., pp. 65-67.

9. Cole, J.; International Lead Zinc Research Organization, New York City; Testimony presented to EPA, Dallas, Texas, April 28, 1972.

10. Airborne Lead in Perspective, op.cit., pp. 65-67.

11. Rabinowitz, M.A., G.W. Wetherill, and J.D. Kopple, "Study of lead metabolism using stable isotope tracers," paper presented at EPA-NIEHS Conference on Low Level Lead Toxicity, Raleigh, N.C., Oct 1-2, 1973 and M. B. Rabinowitz, G. W. Wetherill and J. D. Kopple, "Lead Metabolism in the Normal Human: Stable Isotope Studies," Science 182:725-727, November 1973.

12. Kehoe, R.A.; The Metabolism of Lead in Man in Health and Disease. The Harben Lectures, 1960. McCorquodale and Co., Ltd., London, England, 1961. 81 pages. Reprinted from the J. Roy. Inst. Public Health Hyg.

24:81-97, 101-120, 129-143, 177-203; 1961.

13. Joint FAO/WHO Expert Committee on Food Additives, op.cit.

14. Airborne Lead in Perspective, op.cit., p. 110.

15. Airborne Lead in Perspective, op.cit., p. 51.

16. Shearer, S.D., EPA; Testimony presented at EPA Public Hearings on Regulation of Fuels and Fuel Additives, Los Angeles, California, May 2-4, 1972.

17. Knelson, J.H., R.J. Johnson, F. Coulston, L. Goldberg and T. Griffin; "Kinetics of Respiratory Lead Intake in Humans:" Proceedings of the International Symposium on Environmental Health Aspects of Lead;" CEC, CID, Luxembourg, May 1973, pp. 391-401.

18. Knelson, J.H., F. Coulston, L. Goldberg and T. Griffin; The Role of Clinical Research in Establishing Standards for Atmospheric Lead. Presented at the Conference on Heavy Metals, VDI-Kommission Reinhaltung der Luft, Dusseldorf, Germany, February 22, 1973.

19. Goyer, R.A.; "Lead and the Kidney" in Current Topics in Pathology, Springer-Verlag, Berlin, 55:147-176, 1971.

20. Tepper, L.B. and L.S. Levin; A survey of Air and Population Lead Levels in Selected American Communities. Department of Environmental Health, College of Medicine, University of Cincinnati, Cincinnati, Ohio. Final Report (Contract PH-22-68-28), December 1972. 73 pages.

21. Hasselblad, V. and W.C. Nelson; Additional Analyses of the Seven City Lead Study. Human Studies Laboratory, National Environmental Research Center, U.S. Environmental Protection Agency, Research Triangle Park, N.C. In-house Technical Report, March 15, 1973.

22. Daines, R.H., D.W. Smith, A.F. Feliciano and J.R. Trout; Air Levels of Lead Inside and Outside Homes. Ind. Med. Journal 41:26-28, October 1972.

23. Airborne Lead in Perspective, op.cit., p. 29.

24. Creason, J.P., O. McNulty, L.T. Heiderscheit, D.H. Swanson and R.W. Buechley; Roadside Gradients in Atmospheric Concentrations of Cadmium, Lead and Zinc. Trace Substances in Environmental Health V. A Symposium, 1972. pp. 129-142.

25. Helena Valley, Montana, Area Environmental Pollution

Study, U.S. Environmental Protection Agency, Office of Air Programs, Research Triangle Park, N.C., January 1972. OAP No. AP-91, xiv + 179 pages.

26. Trout, J.R.; Statistics Center, Rutgers University, New Brunswick, N.J. 08903. Personal communication to Dr. K. Bridbord, U.S. ENvironmental Protection Agency, March 21, 1973.

27. Azar, A., K. Habibi and R. Snee; Relationship of Community Levels of Air Lead and Indices of Lead Absorption; Proceedings of an International Symposium on Environmental Health Aspects of Lead; CEC, CID, Luxembourg, May 1973, pp.581-594.

28. Ferrand, E.F., Assistant Commissioner for Scientific Affairs, New York City Department of Air Resources, New York City, New York. Testimony submitted to EPA, April 11, 1972.

29. Jones, R.D., B.T. Commins and A.A. Cernik; Blood Lead and Carboxyhemoglobin Levels in London Taxi Drivers. Lancet, 302-303, August 12, 1972.

30. Hammer, D.I., J.F. Finklea, R.H. Hendricks, T.A. Hinners and W.B. Riggan; Trace Metals in Human Hair as

a Simple Epidemiologic Monitor of Environmental Exposure. Trace Substances in Environmental Health V. A Symposium. D.D. Hemphill, Ed. University of Missouri, Columbia, 1972. pp. 25-38.

31. Nordman, C.H., S. Hernberg, J. Nikkanen and A. Ryhanen; Blood Lead Levels and Erythrocyte Delta-Aminolevulinic Acid Dehydratase Activity in People Living Around a Secondary Lead Smelter. Work - Environm. - Hlth 10:19-25, 1973.

32. King, B.G.; Maximum Daily Intake of Lead Without Excessive Body Lead Burden in Children. Amer. J. Dis. Child. 122:337-339, 1971.

33. Ibid.

VI. Lead Exposure from Dustfall

Recent surveys of urban environments reveal substantial levels of lead contamination in dust and dirt found in streets as well as in homes. This contamination represents a potential source of lead exposure, especially for children. Weathering of leaded paint from buildings is a major contributor to lead in dust and dirt (1-4), as is fallout of airborne lead from other stationary and mobile lead sources. At the moment it is not possible to give an overall figure quantifying the contribution that each of these sources makes to lead in dirt and dust. The discussion to follow summarizes what is known about these contributions as well as the implications of this contamination.

Lead content of dirt at various distances from houses and roadways in Detroit was examined by Ter Haar, et.al. (5). Lead content of dirt two feet from painted frame houses averaged 2,010 ppm lead, while, dirt two feet from brick houses with painted trim averaged 468 ppm lead. Street gutter dirt near these homes averaged 966 and 1,213 ppm lead, respectively. Lead content of dirt decreased with increased distances from both roadways and houses. Lead concentrations in dirt at corresponding distances from frame houses were similar in city and rural yards. Based upon these findings, it was concluded by Ter Haar that lead antiknocks did not contribute significantly to the lead content

of dirt in urban areas. (5) This conclusion, however, does not adequately reflect the effect of proximity to roadways upon dirt lead content that was found. Further, these observations were made in areas with relatively low traffic density and would not be applicable to all urban situations.

In this context, it has been shown that airborne lead fallout decreases with increased distances from roadways (6), implicating lead in gasoline as a contributor to this problem. The effect of proximity to roadways upon lead levels in soils has previously been demonstrated (7). Lead levels in dust samples taken inside homes located within 5 meters from heavily travelled roadways averaged nearly 1000 ppm (0.1%) lead, or approximately double the level in samples from homes located on side streets (8).

Lead concentrations in surface soils of city parks have been found to range from 194 to 3,357 ppm and lead levels in dust from residential and commercial sites in midwestern cities averaged, 1,636 and 2,413 ppm, respectively (9). Dust lead levels in street sweepings from Washington and Boston usually ranged from 0.1 to 0.25% and at times exceeded 0.5% (10,11).

Lead levels in dust inside homes of Boston have ranged from 0.1-0.2% (12) and have exceeded 0.5% in Birmingham, England (13). Samples of dust from inside elementary school classrooms in

Philadelphia and dirt from playgrounds adjacent to those schools routinely contained 0.2-0.3% lead, with levels greater than 1% found at two locations (14). From these studies it is not possible to determine the relative contribution that erosion of lead based paint compared to airborne sources made to lead in housedusts. In some of the measurements in Philadelphia, contamination by lead fallout from industrial sources was believed to have occurred.

Concentrations of lead in house dust have been observed to vary with the amount of lead fallout from the air (15). House dust lead levels taken from middle income homes in two New York City communities which were likely free of peeling lead paint averaged 608.6 ppm and 741.5 ppm, respectively. These values were 2-3 times higher than the mean values from homes in a control community where dustfall lead was about 1/10 - 1/5 as great. In this study, lead in housedust as well as lead in soil from each community followed the dustfall gradient implicating airborne sources largely due to lead in gasoline as important contributors to this contamination. Erosion of lead based paint could, of course, also have been a contributing factor. Lead concentrations in hair among subjects living in these homes (adults and children combined) were not found to consistently follow these exposure differences. However, additional analyses are necessary to determine if lead exposure was different depending on age within each area and if hair lead levels

reflected exposure differences among the young children in different areas. It should be noted that hair leads among children in this study may not strongly reflect housedust lead exposure differences since opportunities to place dust contaminated objects into their mouths during unsupervised activities may be much lower for middle income than for low income children.

To date, ingestion of lead-contaminated dirt or dust has rarely been suspected of causing clinical lead poisoning, but this source of lead generally has not been investigated in cases of excessive lead exposure. A report from Charleston, South Carolina has shown that soil from yards of homes where cases of pediatric lead poisoning occurred contained greater concentrations of lead than soil in yards of randomly selected homes (16). The cases of lead poisoning also were most frequent in areas of the city with high soil lead concentrations. A quantitative assessment of the sources responsible for high soil lead concentrations in Charleston is not possible from this study. In particular, weathering of outdoor paint as well as ashes, pesticides and automotive exhausts were all believed to have contributed. This study concluded that " . . . these facts further support the theory that children in Charleston can get lead poisoning by eating dirt."

In contrast to these findings, a study from England has shown that average blood lead levels in children residing in an area with soil containing 10,000 ppm lead were only 4 µg/100 ml higher than in children from a community with soil containing 500 ppm lead (17). Why children in Charleston, but not in England, appeared significantly affected by residence in areas with high soil lead content is not certain. Perhaps this difference was due to a greater incidence of pica among children in Charleston or the more frequent presence of additional sources of lead in Charleston such as leaded paint. Differences in biologic availability of lead bound to soil in England compared to Charleston may also be important. Lead in dirt and dust which is not tightly bound to soil likely has greater biologic availability than lead which is bound to soil. It should also be noted that lead in the form of fine dusts may be more readily absorbed from the gastrointestinal tract than comparable quantities of lead in paint chips due to the vastly increased surface area in these fine dusts.

In this context, rats fed lead-contaminated top soil gathered near a lead smelter in El Paso at a dose of 1mg lead/day for 28 days, showed blood lead increases of 20 µg/100ml over controls on a low lead diet (18). Similar results were seen after feeding rats lead contaminated dust (1-2% lead) from city highways and automobile tunnels (18). Lead contaminated dirt (6600 ppm) from expressways in Chicago has also been noted to increase blood and

soft tissue lead content after being fed to rats (19). These results suggest that lead in dust, both that gathered near the lead smelter and that originating from emissions of automobiles burning lead additives, although of different chemical composition and perhaps somewhat different particle size, is comparably absorbed when fed to experimental animals.

In recognition of the possibility that ingestion of lead-contaminated dirt and dust could contribute to pediatric lead exposure, the National Academy of Sciences Lead Panel concluded (20):

> "The swallowing of as much as 1g of such dust could result in the oral intake of an amount of lead that exceeds by a factor of 10 or more the estimated mean daily intake of lead from normal food and drink in nonexposed young children . . . For a child with pica for paint, the combination of the ingestion of a few chips of paint and an increased intake of lead from contaminated dusts would provide a total lead intake sufficient to cause symptomatic illness."

There are many factors which place young children potentially at a greater risk to lead exposure from dust than adults. The behavioral patterns of children expose them to large amounts of dust. This was demonstrated in a study from El Paso, Texas (21). In a neighborhood near a lead smelter, with high concentrations

of lead in air (5µg/m^3) and in surface soil (0.1-0.5%), blood lead levels of both children and adults were found to be elevated. Air lead, blood lead and surface soil lead concentrations all decreased with increased distance from the smelter implicating this source as the probable causal factor. Under these circumstances, 80% of the children between 1 and 4 years of age had blood lead levels in excess of 40µg/100g, a frequency much higher than seen among older children and adults. This suggests a special exposure route for young children, not only in dry areas where little or no vegetation exists to hold outdoor dust in place, but also in typical urban situations (25, 38, 43).

Preliminary data suggest that airborne lead concentrations tend to be greatest at ground level, and to decrease with increased elevation (8,22). Therefore, young children, with their shorter height, may be exposed to greater amounts of airborne lead and lead-contaminated dust than older, taller individuals. The normal play activities of children can include placing materials or their hands, contaminated by leaded dusts, into their mouths (23-25). A moistened lollipop dropped to the ground can pick up 0.5g of dirt (22), which would contain 2,500µg of lead if the dirt contained 0.5% lead. This quantity of lead is an order of magnitude greater than the amount of lead routinely found in the daily diet of children.

A Rochester, New York study has demonstrated that lead contamination on hands of young children living in homes with high housedust lead levels is significantly greater than on hands of children living in homes with low house dust lead levels (25). The lead level in household dust was also significantly elevated in inner city homes where at least one child with moderately elevated blood lead resided compared to suburban homes. Since blood leads were not determined in all subjects, it is not known with certainty whether more lead on the hands was associated with increased blood leads in these children. These data are consistent with the frequent contamination of finger prick blood lead specimens by lead on hands of children indicating the widespread occurrence of such contamination.(25)

These data do not firmly establish the sources responsible for the elevated lead concentrations in urban housedust. Of note is that pre-1940 homes had greater quantities of lead in housedust than post-1940 homes (25). This observation was also made in Boston(11). Though this difference could in theory be accounted for by higher concentrations of lead in paint in older homes, it could also be explained by greater ventilation and increased lead fallout in older homes especially since newer homes are more frequently air conditioned (11). Peeling lead paint was likely not the sole contributor to lead in housedust in the Rochester homes studied since an association was not found between the presence of peeling lead based paint in homes and

increased lead in house dust. This in part could have been due to residual leaded dusts from paint which were ground into the floor prior to repainting of deteriorating surfaces or from deterioration of repainted surfaces themselves. With the available data, one cannot quantitatively assess the relative contribution of erosion of lead paint and of other lead sources to the total lead content of house dust. As noted earlier, lead in housedust is a function of airborne lead fallout and proximity of homes to roadways.

In separate Rochester surveys (25) average blood leads among 3-4 year old urban children (about 40µg/100g) were approximately double those among suburban children. Blood leads much above 60 µg/100 g were not common in the urban children, which would be expected if ingestion of paint flakes containing very high quantities of lead were the primary mechanism of exposure. Nearly 50% of these inner city children had blood lead levels between 40 and 60µg/100g implicating sources of exposure less massive than that routinely found in chips of paint. There were no known industrial sources of lead pollution in the inner city study area and high lead levels in water were also ruled out as an important factor. To the extent that airborne lead fallout, in large part due to automobiles, is greater in urban compared to suburban areas, lead in gasoline would have contributed to lead in housedust found in these Rochester homes. Traffic density in this study area was relatively low so the contribution of lead

from gasoline was no doubt less than in areas with high traffic density. These data suggest that ingestion of leaded dusts were in part responsible for increased lead exposure among Rochester children. This study does not quantify the contributors to lead in housedust whether from airborne sources, chalking or peeling of paint, or perhaps pulverization underfoot of paint chips shed from the walls.

Lead-210, an isotope usually present at very low levels in leaded paint and in significantly higher concentrations in fallout dust was employed to evaluate the relative quantities of lead containing dust and paint ingested by young children (5). The hypothesis examined was that if a child with an elevated lead body burden had high stable lead but normal level-210 in his feces, then lead elevation was probably the result of paint ingestion. If high stable lead and high lead-210 were found in the feces, then this would favor lead in dust and dirt as contributing to the lead body burden.

Eight children suspected of having elevated lead body burdens were compared to ten children living in good housing where lead poisoning was not a problem. Fecal excretion of both stable lead and lead-210 was determined in both groups of children. The children suspected of having elevated lead body burdens, with two exceptions who were normal, had stable lead concentrations in their feces that were 4 to 400 times higher than found in the

normal children. There was, however, no difference in lead-210 fecal excretion between groups. Based upon these findings, it was concluded that children suspected of elevated lead body burdens did not ingest dust.

The effect that lead-210 in the diet or in the air could have had upon these results was not adequately considered in this study. Unless lead-210 intake from the diet and the air were known to be equivalent in each group, no firm conclusions are warranted regarding non-dietary sources of lead-210. Unfortunately, no measurements of lead-210 in the diet were made, which for adults in the United States range from 1-2 picocuries/day (26-28). Airborne sources which have been estimated to contribute 8-32% (28) and in one instance nearly 50% (27) of total daily lead-210 absorption into the blood were also not measured. From the data presented (5), one can calculate that the fecal excretion of lead-210 by these children was generally less than 1 picocurie/day. Except for one child who almost certainly had recently ingested paint chips, fecal stable lead excretion in the group suspected of elevated lead body burdens averaged only about 300 µg/day or 5 times greater than in the normal children. This level of lead excretion is far below the 44,000 µg/day associated with lead poisoning from ingestion of paint chips (2). Though these findings do not rule out ingestion of paint as dust or ingestion of paint chips prior to fecal lead determinations in these children, they also do not

rule out ingestion of dirt or dust contaminated by lead sources other than paint prior to examination.

An additional important observation is evident from this study. The lead-210 which was used as a tracer is a product of radon decay in the atmosphere. Lead-210, thus produced, attaches to air suspended particulates including lead from automobile exhausts as well as from other sources. The fact that relatively high concentrations of lead-210 originating in the atmosphere were found in vacuum cleaner sweepings, in yard dirt and in street dirt means that atmospheric particulate fallout, known to routinely contain stable lead, was contaminating these samples to some degree.

Extensive screening programs in the United States concentrating on inner city children, have demonstrated that approximately 25% of these children were significantly exposed to lead (blood lead levels of 40µg/100g or above) (29-31). Lead-based paint has long been recognized as the principle source of lead overexposure among young children. However, it is not clear that lead-based paint alone accounts for this widespread degree of exposure.

For example, screening programs have detected high levels of lead exposure in children residing outside the central city, where exposure to lead based paint would be expected to be less

than in the central city environment (24,32,33). In a study from Oregon, 50% of children with elevated blood lead levels were found to live in housing where lead paint was not considered accessible to them (34). In New York City, only 50% of children with blood lead levels between 35 and 44µg/ 100g could be associated with living in homes containing peeling paint of 1% or greater lead. Nearly 20% of this group lived in homes in which no peeling paint was identified (35).

The source of excessive lead exposure is thus not always directly related to paint in a significant percentage of cases. This situation may reflect exposure to lead paint in former homes, or in places other than those inspected. Further, closer or repeat inspection will often reveal sources of peeling lead paint in the home which were not initially detected. Additional sources of lead in food, water, pottery, toys, pencils, solder, etc., are also possible factors in cases of unidentified routes of exposure. Lead contaminanted dust and dirt, especially prevalent in urban areas, may also be an important contributing factor. Further information is required to arrive at a definitive assessment of the significance of this source of lead contamination.

It is clear that peeling lead based paint remains an exceedingly important problem, and even exists outside of inner city areas (36). In one New York City study, 100% of apartments

in a high risk lead poisoning neighborhood showed high lead levels on paint surfaces (37). These studies, and extensive clinical experience, indicate that leaded paint is primarily responsible for the great majority of overt clinical lead poisoning in children, although non-paint lead sources would decrease the amount of lead from paint required to cause serious damage. Education and screening programs have been noted to decrease the reported frequency of clinical lead poisoning in children (38). Whether or not these screening programs are sufficient in themselves to adequately reduce the frequency of undue lead exposure among children is not certain.

For example, in the initial year of the Chicago lead screening program, 8.5% of children tested had blood leads of 50 µg/100 ml or more (38). In 1968 the fraction of children with blood leads in this range dropped to 3.8% and in 1969 to 1.5%. Figures for 1970 and 1971 were 2.1% and 2.3% respectively. Hence, despite an initial decline and a continued and vigorous program to decrease lead paint exposure, the percentage of children tested with blood leads of 50 µg/100 ml or above has either remained relatively constant or may have actually increased slightly in recent years. Statistics for 1972 confirm this increasing trend and show that 4.1% of children screened that year had blood lead levels of 50 µg/100 ml or above (42). Thus, though efforts to reduce lead paint exposure continued, the reported frequency of undue lead exposure but not severe lead

poisoning appears to be increasing in Chicago. These findings are unexplained by changes in method of blood lead collection or analytical techniques because these procedures have not been altered since the beginning of the Chicago screening program in 1967. This increasing trend is particularly significant since screening programs initially concentrate in highest risk areas for lead paint and then at least to some degree gradually expand to include other areas of lesser risk. These statistics indicate the continuing need for vigorous action to prevent lead paint exposure and also suggest that sources of lead other than paint are having an incremental effect upon lead paint exposure in Chicago.

There has admittedly been considerable controversy as to whether or not partial or even total removal of lead from gasoline would have any impact upon the lead exposure problem in this country (39,40). In this context, children residing in pre-World War II housing have been observed to have higher blood lead levels than children residing in newer housing projects. One such survey conducted in Washington, D.C. during 1970 indicated that the lowest blood lead levels among children tested for lead poisoning were found in areas of the city where public housing projects had been built since 1942 (43). No child living in a recently built housing project, where exposure to lead based paint would be minimum, had a blood lead level above 40 µg/100g. Blood lead determinations in this survey of 808 children showed

that 5.8% of all children tested had a blood lead of at least 40 µg/100g and that nearly 10% of children between 1-3 years of age had blood lead levels in this range. It should be noted that these results are somewhat lower than those from subsequent Washington, D.C. surveys in which 22.0 and 39.2% of children tested were found to have blood lead levels of 40 µg/100g or above (44,45).

This original Washington survey does indicate that the frequency of lead poisoning and undue lead absorption in children would be greatly reduced if lead paint were nearly eliminated. These findings however, do not rule out an important contributing role for sources of lead other than paint among children residing in home environments where lead paint is present.

For example, location of residences near urban roadways and in areas of high traffic density has been shown to significantly affect the degree of undue lead absorption among children also exposed to paint (46). In this study, the effect of residential location upon blood lead was examined retrospectively among over 5,000 children tested for lead poisoning in Newark, New Jersey during 1971. Among those children residing within 100 feet of a major roadway, 49.3% were found to have blood lead levels between 40-59 µg/100 ml and 8.1% had blood lead levels of 60 µg/100 ml and above. Children residing 100-200 feet and greater than 200 feet from major roadways were not significantly different from

each other. Approximately 25% of blood leads in both groups were between 40-59 µg/100 ml and 3-5% were at least as high as 60 µg/100 ml. When these groups, however, were compared to children residing within 100 feet of major traffic arterials a marked difference was observed with twice the frequency of undue lead absorption found in children living nearest the roadway.

In children residing within 200 feet of a major traffic artery the extent of undue lead absorption was observed to increase when average weekday traffic densities exceeded 24,000 vehicles per day. In such instances 51.3% of children had blood leads between 40-59 µg/100 ml and 10.8% of children had blood leads of 60 µg/100 ml or above. Comparable rates for children residing in areas with weekday traffic densities below 24,000 vehicles per day were 36.6% with blood leads between 40-59 µg/100 ml and 5.1% with blood leads of at least 60 µg/100 ml.

These results could conceivably be confounded by existence of poorer quality housing nearest roadways and by racial and social class differences among residents near major roadways. However, the fact that no difference was noted in blood leads among children residing within 100-200 feet of roadways compared to children residing beyond 200 feet suggests that these other factors were likely not major confounding variables.

Earlier studies in Newark found positive and statistically significant, but not overwhelming correlations, between elevated blood leads and indices of housing status as reflected by number of housing units built prior to 1950 and the number of housing units considered deteriorated and dilapidated in the census tract. (47) Correlation coefficients for these indices and rates of excessive lead absorption (blood lead of 60 µg/100g and above) and elevated blood lead (40 µg/100g and above) ranged from 0.35 to 0.49. The relatively low correlations which were observed suggests that factors other than housing status also were important. For example, in seven census tracts with low potential hazard scores based on housing status, high rates of excessive lead absorption were observed.

Findings such as these from Newark indicate that factors other than housing are important in lead poisoning and that automotive lead emissions can contribute significantly to undue lead absorption in children. Because immediate removal of old lead paint from all existing structures cannot practically be accomplished, reduction of other sources of lead exposure is important. If the results from Newark, New Jersey are in any way applicable to other urban situations, this would indicate that automotive lead emissions do have a significant incremental impact upon the lead exposure problem in this country.

In a study in Philadelphia, referred to earlier (14), 8.4% of white first grade children who lived and attended private school in proximity to a major industrial lead source were found to have dentine lead levels in excess of 300 µg/g, a level associated with frank lead poisoning. These children resided in older housing that was generally in good repair but were exposed to levels of lead in environmental dusts commonly in the range of 3,000-8,000 ppm. The community in which these children lived was also heavily exposed to automotive lead emissions. In contrast to these children, 19% of black students attending public school in the same district but who lived in deteriorating housing had dentine lead levels in excess of 300 ug/g. When both groups of children were compared to public and private school children living in a community free of industrial lead sources and residing in housing built since World War II striking differences in dentine lead levels were noted. Only 3% of the public and 6.6% of the private school students in the low exposure community had dentine lead concentrations in excess of 100 µg/g, whereas 66% of the black public school students and 43% of the white private school students in the high exposure area had dentine lead levels greater than 100 µg/g.

Interior dust samples from the public and private schools in the low exposure community contained an average of 614 ppm lead (range 293-939 ppm) compared to interior dust samples from schools in the high exposure community which contained an average

of 3,867 ppm lead (range 929-15,680 ppm). Samples of gutter dirt in the low exposure community contained an average of 1,507 ppm lead (range 270-2,626 ppm) compared to an average of 3,262 ppm lead (range 280-8,201 ppm) for gutter dirt samples in the high exposure community. These data from Philadelphia are suggestive that airborne and dust lead largely from industrial sources can contribute to undue lead absorption in typical urban areas either by themselves or when combined with paint from deteriorating housing.

Studies from Philadelphia, Chicago and Newark discussed above provide persuasive evidence to strongly suggest that sources of lead other than paint, including that resulting from the presence of lead in gasoline, play an important role in childhood lead exposure. These other sources may be especially significant at levels of exposure below overt clinical poisoning.

The National Academy of Sciences summarized available information on the ingestion of lead by children in the following paragraph from its (1972) report (41):

> "The extent to which airborne lead in congested urban areas contributes to increased lead absorption and lead poisoning in children is not clearly defined. With respect to young children, it is not a matter exclusively of inhalation and particle size, inasmuch as young children mouth and actually

eat things that are not food rather indiscriminately. Airborne lead wastes from such sources as automotive emissions and the weathering and demolition of old buildings can be expected to have a significant additive effect on the total intake. This would be sufficient to evoke compensatory metabolic responses that are now considered subclinical (such as increased urinary ALA), at the very least. It may be estimated that dustfall from airborne lead, if swallowed, can make a significant contribution to a small child's total lead intake and thereby contribute to the occurrence of lead poisoning, especially in urban areas. Even so, the direct ingestion of lead-pigment paints is clearly the principal environmental source in cases of severe acute lead poisoning in young children."

In conclusion, clinical experience indicates that leaded paint is primarily responsible for the great majority of overt clinical lead toxicity in children. There is, however, sufficient data to strongly suggest that sources of lead other than paint play an important role in childhood lead exposure. These other sources may be especially significant at levels of exposure below overt clinical poisoning.

Lead in the air, and particularly lead in dust are ubiquitous sources of lead which may well be important contributing factors to the problem. Exposure to dirt and dust sufficiently

contaminated by lead could reduce significantly the quantity of additional lead exposure required to produce clinical poisoning in a child with other sources of exposure. Though exposure to lead contaminated dirt and dust from automobile exhaust, alone, has not been shown to be responsible for cases of overt lead poisoning, automotive lead has been related to undue lead absorption in children. At this time, it would be prudent to decrease the potential air and dust lead exposure. It should be recognized that further studies are necessary to better quantitate the sources of lead contamination in dust and dirt and the magnitude of the contribution that leaded dust and dirt make to both subclinical and clinical lead overexposure.

REFERENCES

1. Hardy, H., et al., "Lead as an Environmental Poison," Clinical Pharm. and Ther., 12:982-1002, November 1971

2. Comments submitted by Ethyl Corporation to the Environmental Protection Agency, March 9, 1973. pp. V-14 - V-19

3. De Treville, T.P., "Natural Occurrence of Lead," Arch. Env. Health, 8:212-221, February 1964

4. Bertinuson, J.K. and Clark, C.S., "The Contribution of Lead Content of Soils from Urban Housing," Interface, 6: p. 1073, 1973

5. Ter Haar, G. and R. Aronow, "New Information on Lead in Dirt and Dust as Related to the Childhood Lead Problem," paper presented at EPA-NIEHS Conference on Low Level Lead Toxicity, Raleigh, N.C., Oct 1-2, 1973

6. Creason, J.P., O. McNutty, L.T. Heiderscheit, D.H. Swanson, and R.W. Buechley; "Roadside Gradients in

Atmospheric Concentrations of Cadmium, Lead and Zinc," Trace Substances in Environmental Health V. A Symposium, 1972 pp. 129-142

7. Airborne Lead in Perspective, A report prepared by the Committee on Biologic Effects of Atmospheric Pollutants, National Research Council, National Academy of Sciences, Washington, D.C. 1972, p.29

8. Darrow, D.K. and Schroeder, H.A., "Childhood Exposure to Enviromental Lead," paper presented before the American Chemical Society, Chicago, Ill., Aug. 29, 1973

9. Airborne Lead in Perspective, op. cit., p. 30

10. Fritsch, A. and M. Prival, Center for Science in the Public Interest, Washington, D.C., testimony submitted to EPA, 1972

11. Krueger, H. Krueger Enterprises, Cambridge, Massachusetts, testimony submitted to EPA, July 10, 1972

12. Krueger, H. Op. cit.

13. Personal communication to L. Lombardo, Public Interest Campaign, from R. Stephens and A. Townshend, University of Birmingham, Department of Chemistry, Birmingham, England, January 8, 1973

14. Personal communication from Dr. I. M. Shapiro, U. of Penn. School of Dental Medicine, Philadelphia, Penn. to Dr. K. Bridbord, EPA, July 26, 1973 and H. L. Needleman and I. M. Shapiro, "Dentine Lead Levels in Asymptomatic Philadelphia School Children: Subclinical Exposure in High and Low Risk Groups," paper presented at EPA-NIEHS Conference on Low Level Lead Toxicity, Raleigh, North Carolina, October 1-2, 1973.

15. Pinkerton, C., J. P. Creason, D. I. Hammer and A. V. Colucci," Multi-Media Indices of Environmental Trace Metal Exposure in Humans," presented at 2nd International Symposium on Trace Element Metabolism in Animals, June, 1973, Madison, Wisconsin

16. Fairey, F. S., and J. W. Gray, "Soil Lead and Pediatric Lead Poisoning in Charleston, S.C.", J. South Carolina Med. Assoc., 66:79-82, March 1970

17. Barltrop, D., and Strehlow, C.D., "The Significance of High Soil Lead Concentrations in Childhood Lead Burdens,"

paper presented at EPA-NIEHS conference on Low Level Lead Toxicity, Raleigh, N.C., Oct 1-2, 1973

18. "Environmental Lead: Comparison of Blood and Tissue Levels Following Oral Exposure in Rats," EPA Internal Report, ETRL, NERC-Cincinnati, February, 1973

19. Calandra, J.C., Industrial Bio-Test laboratory, Northbrook Ill., Industrial Bio-Test Research Report IBT No. E1733C, January 10, 1973

20. Airborne Lead in Perspective, op. cit. p. 139

21. Data compiled by Center for Disease Control, HEW, Atlanta, on human and environmental lead pollution in El Paso, Texas, 1972

22. Personal communication from Dan K. Darrow, Dartmouth Medical School, Trace Element Laboratory, Brattleboro, Vt. to R. K. Bridbord, EPA, Washington, D.C., February 20, 1973

23. Barltrop, D., "Children and Environmental Lead," paper presented at a conference on "Lead in the Environment," Zoological Society of London, January 27, 1972

24. Lin-Fu, J. S., "Preventing Lead Poisoning in Children," Children Today, Vol. 2, p2, January 1973

25. Sayre, J.W., Vostal, J., Pless, I.B., and E. Charney, House and Handdust as a Potential Source of Childhood Lead Exposure, Am. J. Dis Child, in press, 1973 and Vostal, J., J.W. Sayre, E. Taves and E. Charney, "Lead Containing House Dust: another Source of Increased Lead Exposure in Inner City Children," paper presented at EPA-NIEHS Conference on Low Level Lead Toxicity, Raleigh, North Carolina, October 1-2, 1973

26. Holtzman, R.B., "Measurement of the Natural Contents of RaD (Pb-210) and RaF (Po-210) in Human Bone-Estimates of Whole-Body Burdens", Health Physics 9:pp 385-400, 1963

27. Morse, R.S. and Welford G.A., "Dietary Intake of 210-Pb," Health Physics, 21:pp. 53-55, 1971

28. Magno, P.J., Groulx, P.R. and Apidianakis, J.C., "Lead 210 in Air and Total Diets in the United States During 1966," Health Physics, 18: pp. 383-388, 1970.

29. Lin-Fu, J.S., "Undue Absorpticn of Lead Among Children--A New Look at an Old Problem," New Eng. J.

of Med., Vol. 286, pp. 702-710, 1972

30. Fine, P.R., G.W. Thomas, R.H. Suhs, R.E. Cohnberg and B.A. Flashner, "Pediatric Blood Lead Levels, A Study in 14 Illinois Cities of Intermediate Population," JAMA: 221, p. 1475-1479, September 1972

31. Gilsinn, J.F., "Estimates of the Nature and Extent of Lead Paint Poisoning in the United States," NBS Technical Note 746, December 1972

32. Lin-Fu, J.S., "Vulnerability of Children to Lead Exposure & Toxicity," New England Journal of Medicine, In Press

33. Personal communication from W. J. Sobolesky, City of Philadelphia Department of Public Health to K. Bridbord, EPA, dated August 6, 1973

34. Personal communication from M. Deas, Department of Human Services, Multnomah County, Oregon, to K. Bridbord, EPA, dated March 6 , 1973

35. New York City Bureau of Lead Poisoning Control, data submitted to EPA, August 31, 1972 and September 12, 1972

36. Personal Communication from W. R. Smith, Baltimore City Health Department, Baltimore, Md., to M. Kanarek, EPA, Washington, D.C.

37. Lauer, G.R., R.E. Albert, T.J. Knelp, B. Pasternak, C. Strehlow, N. Nelson and F.S. Kent, "The Distribution of Lead Paint in New York City Tenement Buildings," Amer. J. Pub. Health, 63: 163-168, February 1973

38. Sachs, H.K., "Effects of a Screening Program on Changing Patterns of Lead Poisoning," Paper presented at EPA-NIEHS Conference on Low Level Lead Toxicity, Raleigh, North Carolina, October 1-2, 1973.

39. Personal communication from J. J. Chisolm, John Hopkins Univ. School of Medicine, Baltimore, Md. to Asst. Administrator for Air and Water Programs, EPA, Washington, D.C., dated March 6, 1973

40. Personal communications from H. Sachs, formerly Director, Chicago Lead Poisoning Program to Administrator, EPA, dated March 5, 1973 and June 15, 1973

41. "Airborne Lead in Perspective," op. cit., p. 140

42. Personal communication from H. L. Slutsky, Coordinator, Chicago Lead Poisoning Program to J. S. Lin-Fu, HEW, August 28, 1973.

43. Conn, R. H. and D. Anderson, "D. C. Mounts Unfunded Program of Screening for Lead Poisoning," HSMHA Health Reports, 86:409-413, May 1971.

44. Lin-Fu, J. S., Undue Absorption of Lead among Children. A New Look at an Old Problem, New Eng. J. Med., 286:702-710, 1972.

45. Gilsinn, J.F., Estimates of the Nature and Extent of Lead Paint Poisoning in the United States, National Bureau of Standards Technical Note 746, December 1972.

46. Margulis, H. L., R. J. Caprio and M. K. Joselwo, Residential Location Ambient Air Lead Pollution and Childhood Lead Poisoning, Arch. Env. Health, in Press.

47. Margulis, H. L., R. J. Caprio, A. Stefaniwsky, and M. K. Joselow, "Surveillance of Lead Poisoning in Newark, New Jersey--An Ecological Approach,"

A 230

Rutgers University and the New Jersey College of Medicine and Dentistry, Newark, New Jersey. Prepublication draft.

VII. Extent of Lead Exposure Among the General Population

In section IV, it was stated that determination of lead in blood is at present the best index for evaluating lead body burden and exposure. However due to limitations of the studies on blood lead levels in population groups in this and other countries, it is not possible to make precise estimates of the number of persons having blood lead levels within a given range. Problems still exist in the chemical analysis of blood for lead content. The results of quality control studies from laboratories in both the United States and Europe show that sometimes there is a large variation in results among different laboratories (1). Reproducible results even within the same laboratory are not always obtained, as shown in the Seven Cities Study (2). It is difficult to accurately estimate the relative contributions of air and food sources to such blood lead levels as are found. That air lead makes some contribution is apparent from occupational data and from experimental exposures. The limited survey data available make it clear that most of the adult U.S. population has blood lead levels in the range of 10-30 µg/100 g.

Available information on the extent of elevated blood lead levels found in this country is presented in Tables VII-1 & 2. It can be seen that a small but generally consistent proportion of the general population has elevated blood lead levels. It is also evident that a number of specific groups in the population frequently or characteristically have blood lead levels

distinctly higher than those usually found. Many of these groups are occupationally exposed to automobile exhaust. Individuals so exposed in an outdoor environment, such as traffic police, parking lot attendants, and service station attendants frequently show elevated mean blood levels with ranges of 20-50µg/100g or higher (3,4,6-9). The few data on commercial drivers(4,6,7) also show elevations compared to the general population. Likewise, the available information on groups exposed to automotive exhaust in closed environments show consistently elevated blood leads (4,8,9) (See Table VII-1). These include workers in parking structures, traffic tunnels, and service and repair garages. Repairmen and mechanics may, of course, also be exposed to lead dust and fumes originating from maintenance activities (9).

Blood lead levels found in urban areas are also somewhat higher than in suburban and rural areas(2-4) (See Table VII-2). The source of most lead for the general population is food, although air is a significant source of lead in urban areas. It is known that air lead exposures are higher in urban than in suburban and rural areas. There are no data available to determine whether or not this is also true for food. The extent of the contribution, by occupational and avocational lead exposure, to the lead burden of the general population and to urban-rural gradients is likewise not well documented.

In recent years it has been found that large numbers of central city children are overexposed to lead. Surveys have been carried out in urban areas to search for childhood lead

pcisoning, which usually results from the ingestion of chips of peeling lead paint. Other evidence of overexposure was also found. In nearly all of these surveys, it has been demonstrated that approximately 25% of the young children tested have blood lead levels of 40 µg/100g or above (most below 50 µg/100g) (10-13) (Table VII-3), which is medically undesirable. The reasons for this frequency of elevated blood leads are not completely understood at present. The shapes of the blood lead frequency distributions suggest that lead sources more homogeneously distributed than peeling paint and containing less lead than routinely found in paint chips are making a major contribution. The rationale for considering lead dusts which originate from automotive exhaust as a contributing factor was discussed in the previous sections.

In summary, it is clear that levels of lead in the blood, indicating elevated exposure, exist to a small but significant extent in the general adult population, and to a very great extent among children. There are also a number of large groups of the population in which undesirable elevated blood lead levels are commonly found directly related to exposure to automobile exhausts. Thus much of the excess exposure to lead in the general population is due at least in part to lead from automotive exhaust.

TABLE VII-1

Extent of Abnormally Elevated Blood Leads
Among Urban Adults

City	Exposure Category	Number Studied	% of Blood Leads Equal to or Greater than 40 µg/100 g
Cincinnati	Post Office Employees (4)	140	2.9
	Firemen (4)	191	3.0
	Service Station Attendants (4)	130	12.3
	Police (4)	40	12.5
	Drivers of Cars(4)	59	15.0
	Parking Attendants (4)	48	44.0
	Garage Mechanics (4)	152	67.0
Los Angeles Area	L.A. Police (4)	155	0.6
	Pasadena Male City Employees (4)	88	3.3
	L.A. Female Aircraft Employees (4)	87	3.3
	L.A. Male Aircraft Employees (4)	291	5.2
Philadelphia	Male Commuters (1)	43	2.3
	Police (4)	113	3.5
	Downtown Male Residents (4)	66	4.5
Camden, New Jersey	Women Living Near Freeways (5)	55	1.8
Composite Urban Samples	Females from New York, Philadelphia, and Chicago (3)	423	0.7
	Males and Females from 6 Cities (3)	833	2.7*
Composite Urban Samples	Taxi Drivers and Office Workers from L.A.; Philadelphia; Barksdale, Wisconsin, and Starke, Florida (6)	149	0

*Only those above 40 µg/100 g blood lead.

TABLE VII-2

Urban-Suburban Blood Lead Comparisons
In Adults

Group Studied	Number Studied	% Blood Leads Equal to or Greater than 40 µg/100 g
Urban Females (2)	423	0.7
Suburban Females	556	0
Phiadelphia Males (4)		
Urban	66	4.5
Suburban	23	0
Composite (3)		
Urban	833	2.7*
Suburban	162	0

*Only those above 40 µg/100g.

TABLE VII-3

Percentages of Children with Abnormally Elevated Blood Leads

City	Years Tested	Number Tested	% Blood Leads Equal to or Greater than 40 µg/100 g
Baltimore (10)	1968	655	25.3
	1969	746	27.9
	1970	939	31.5
Chicago (10)	1967-70	120,000	20.0
New Haven (10)	1969-70	1,897	29.8
Newark (10)	1970	594	38.9
New York (10)	1969	2,648	45.5
	1970	84,368	28.7
New York (11)	1971	81,626	20.2
Philadelphia (10)	1970	3,496	34.0
Washington (10)	1970	808 (all ages)	5.8
	1970	1,152 (2 years)	22.0
Many Cities (12)	1971	2,309	9.1
Aurora, Ill. (13,14)	1971	449	24.3
Springfield, Ill. (13,14)	1971	670	30.1
Peoria, Ill. (13,14)	1971	387	31.3
E. St. Louis, Ill. (13,14)	1971	376	24.7
Decatur, Ill. (13,14)	1971	793	12.2
Joliet, Ill. (13,14)	1971	383	24.3
Rock Island, Ill. (13,14)	1971	285	21.1
E. Moline, Ill. (13,14)	1971	298	11.4
Robbins, Ill. (13,14)	1971	103	12.6
Harvey, Ill. (13,14)	1971	226	16.4
Carbondale, Ill. (13,14)	1971	264	17.0
Norfolk, Va. (13)	1971	1,225	22.7
New Haven, Conn. (13)	NA	1,339	23.7
Washington, D. C. (13)	1971	1,821	39.2
Rockford, Ill. (13,14)	NA	1,200	19.5

REFERENCES

1. Subclinical Lead Poisoning; Editorial; Lancet; 1:87, January 1973

2. Tepper, L. and L.S. Levin, "A Survey of Air and Population Lead Levels in Selected American Communities"; Department of Environmental Health College of Medicine, Univ. of Cincinnati, Cincinnati, Ohio; Final report (Contract PH-22-68-28), Dec., 1972, Cincinnati, Ohio

3. Hofreuter, D.H.; et.al.; "The Public Health Significance of Atmospheric Lead", Arch. Env. Hlth. 3:82:88, Nov. 1961

4. "Survey of Lead in the Atmosphere of Three Urban Communities", Public Health Service. Publ. No.999-AP-12, 1965

5. Daines, R.H. et. al.; "Air Levels of Lead Inside and Outside Homes"; Indust. Med. 41: 26-28, Oct. 1972.

6. Azar, A., et. al. - Relationship of Community Levels of Air Lead and Indices of Lead Absorption. - pp 581-594 In: Proceedings of International Symposium on Environmental Health Aspects of Lead, Amsterdam, October 2-6, 1972

7. Jones, R.D. et.al. - Blood Lead and Carboxyhemoglobin Levels in London Taxi Drivers - Lancet 2: 302-303, 12 Aug., 1972.

8. Bericht der Eidg. Bleibenzin-Kommission an den Bundesrat uber ihre tatigkeit im zeitraum 1947-1960. Mitt. Gebiete Lebensmitt. Hyg. (Bern). 52(3): 135-244, 1961.

9. Tola, S. et. al. - Occupational Lead Exposure in Finland. II. Service Stations and Garages. - Work Environ. Hlth. 9:102-105, 1972.

10. Lin-Fu, Jane S. - Undue Absorption of Lead Among Children. A New Look at an Old Problem - New Eng. J of Med., 286: 702-710, 1972

11. Guinee, Vicent F. - Lead Poisoning - Amer. Jour. Med.; 52:283-288,

12. Challop, R.S., and McCabe, E.B.; "Childhood Lead Poisoning: A Thirty City Neighborhood Survey"; BCEM, USDHEW, May 23, 1972.

13. Gilsinn, J.F., Estimates of the Nature and Extent of Lead Paint Poisoning in the United States, NBS Technical Note 746, December 1972

14. Fine, P.R., et.al. Pediatric Blood Lead Levels. "A Study in 14 Illinois Cities of Intermediate Population," JAMA 221: p. 1479, September 1972.

VIII. Summary and Conclusions

Summary

Lead occurs widely in the environment. Its present distribution is the result of natural occurrence, greatly influenced by man's activities. In 1971, in the United States, 1,431,514 tons of lead were consumed with over one million tons used as metallic lead or lead alloys and in storage batteries. About 135,000 tons were used in coatings and pigments. Of the 264,000 tons used in gasoline additives, more than two-thirds enters the environment. Combustion of gasoline contributes by far the largest fraction of lead reaching the environment. Other sources of lead in the environment are wear and erosion of lead-containing painted surfaces, and incineration of lead-containing substances.

Man takes in lead from many sources: water; food; air; and, particularly in the case of children, from ingestion of lead-containing non-food items such as paint and dust. It is probable that lead in dust and dirt is inadvertently ingested by both children and adults. It is generally agreed that food is the major source of lead for the general population. A World Health Organization expert committee reports that according to the results of total diet studies in industrialized countries, the total intake of lead from food generally ranges from 200-300 µg per person per day. WHO further states that based upon available

data, these levels are similar to those found in the past 30-40 years and that no upward trend in lead levels in food is evident. Reducing the amount of airborne lead in the environment constitutes an accessible means for reducing potential human exposure to environmental lead particularly when that fraction is large compared with that absorbed from the diet. (Section II)

Lead has not been shown to be biologically essential or beneficial to man. In sufficiently high quantities, it is clearly toxic, and, at somewhat lower levels, has been shown to cause biochemical changes. Lead is also suspected of producing subclinical neurologic damage.

The studies reviewed in Section III permit no unequivocal conclusions to be drawn. On balance, they suggest that subclinical changes are associated with blood lead levels of approximately 40 µg/100g and above. As blood lead levels increase above 40 µg/100g, the likelihood that these changes will occur increases markedly. Based upon evidence from these studies, it would seem prudent to regard blood lead levels over 40 µg/100g as indicators of lead intake that should be prevented. The 40 µg/100g figure, however, does not represent a sharp demarcation between health and disease.

Blood lead levels, under most circumstances, serve as a reasonably accurate measure of lead body burden and have been widely employed in public health surveillance. It is not

possible at this time to firmly establish a single acceptable blood lead level protective of all high risk population groups. It would appear prudent, however, to recommend that the current U.S. Public Health Service Guideline for older children and adults, i.e., 40 µg/100ml whole blood be regarded as a strict upper limit for younger children. Whether the acceptable upper limits should be lower than 40 µg/100ml for the fetus, neonate, and the woman of child bearing age will require further investigation. (Section IV)

In Section V, it is shown that lead from automotive exhaust contributes to increased exposure for humans both from the air and from fallout. Blood lead levels exceeding 40 µg/100g are considered medically undesirable and may ultimately be harmful. Blood lead levels depend on daily dietary intake and adsorption of respired lead. For a "standard man" with average dietary lead intake, exposure to average airborne lead concentrations of 5.0 to 6.7 $\mu g/m^3$ could cause his blood lead concentrations to reach 40 µg/100g within a year. Airborne lead levels near to or in excess of 5 $\mu g/m^3$ have been observed in several U.S. cities.

Lead in dust and dirt is an important potential source of exposure, especially for young children. Daily ingestion of relatively small quantities of dirt or dust (less than 1 gram or about 1/4 of a teaspoonful) containing 1000-2000ppm of lead would be medically undesirable. Although dustfall exposure to lead is still often considered an hypothesis, much of the available evidence is consistent with this hypothesis. Present day lead

exposure via air or dusts in some sections of large cities leaves little or no margin of safety in relation to those concentrations associated with biomedical harm.

In Section VI, it was shown that lead emissions from automobiles contribute to lead present in urban soils, in street dirt and in house dust. Lead content of soil has been demonstrated to decrease with increased distances from both painted houses and roadways. Lead concentrations in fallout dust from the air also decrease away from roadways. Dust lead levels in street sweepings from large cities usually range from 0.1 to 0.25% and at times exceed 0.5%. Housedust from urban areas commonly contains 0.1% lead or higher. Concentrations of lead in housedust vary with lead fallout from the air and are reported to be higher in homes located near heavily travelled roadways than in homes on side streets. Quantities of lead in housedust do not vary directly with the presence of peeling paint in the home but they are higher in older homes, reflecting greater amounts of lead paint, as well as increased atmospheric ventilation and dust fallout in older homes. Erosion of paint is clearly a contributor to lead in housedust. Urban dust and dirt, if sufficiently contaminated with lead represent a hazard to children, if ingested. With the available data one cannot quantitatively assess the relative contribution of erosion of lead paint and of other lead sources to the total lead content of soil, street dirt and housedust. Nevertheless, it is evident that lead emissions from automobiles are major contributors to this contamination.

Clinical experience indicates that leaded paint is primarily responsible for the great majority of overt clinical lead poisoning in children. There is, however, sufficient data to strongly suggest that sources of lead other than paint play an important role in childhood lead exposure. These other sources may be especially significant at levels of exposure below overt clinical poisoning.

Lead in air, and particularly lead in dust are ubiquitous sources of lead, which may well be important contributing factors in the childhood lead problem. Exposure to dirt and dust, if sufficiently contaminated by lead, could significantly reduce the quantity of additional lead required to produce clinical poisoning in a child with other sources of exposure. Although exposure to lead contaminated dirt and dust from automotive exhausts has not been shown to be responsible for cases of overt lead poisoning, automotive lead has been related to undue lead absorption in children. At this time, it would be prudent to decrease the potential air and dust lead exposure. It should be recognized that further studies are necessary to better quantitate the sources of lead contamination in dust and dirt as well as the magnitude of the contribution that leaded dust and dirt make to both subclinical and clinical lead overexposure.

It is clear that undesirable levels of lead in the blood, indicating elevated exposure, and elevated lead body burdens exist to a great extent among children, and to a small, but significant extent among adults. There are also a number of

adult groups in which undesirable elevated blood lead levels are found directly related to exposure to automobile exhausts. Thus, much of the excess exposure to lead in the general population is due at least in part to lead from automotive exhaust. (Section VII)

Conclusions

A small but significant fraction of the adult population has blood lead levels of 40µg/100g or higher, and such levels occur in a much larger proportion of urban children. Such levels are medically undesirable and should be reduced if possible.

Sources of exposure to lead include food, water, air, and ingested non-food items such as lead based paint and dust. Food is the largest contributor of lead to the general population.

Lead based paint is the major cause of overt clinical lead poisoning in children, though sources of lead other than paint play an important role in childhood lead exposure particularly at levels below overt poisoning.

Lead in dust and dirt is believed by EPA to contribute to increased lead levels in man, both through inhalation of resuspended dusts and at least in children, through inadvertent

ingestion of dirt and dust. This source could significantly reduce the quantity of additional lead required to produce clinical poisoning in a child with other sources of exposure. Automotive lead is a major contributor to lead in dust and dirt and has been related to undue lead absorption in children.

Lead from all these sources should be reduced to the degree possible.

Actions have already been taken by the Federal and other levels of government to reduce the lead content of paint and further actions in this regard are being contemplated so that this source of lead will decline with time as older buildings with leaded paint are replaced. Action has also been taken to substantially reduce controllable sources of lead in food, and efforts to further reduce lead in food are continuing. One controllable source of lead in food is residue from use of lead compounds as insecticides. Tolerances for these residues are being reevaluated at this time. Lead in a few drinking water supplies is higher than desirable, and efforts should be continued to reduce this source of lead exposure.

In EPA's opinion, lead in gasoline is the most important remaining source of <u>controllable</u> lead entering the environment. Reduction of lead in gasoline has shown to reduce lead in the ambient air. Leaded gasoline results in direct exposure to the population through inspired air and by presence of lead in dirt

and dusts which may be inspired or inadvertently ingested. General widespread contamination of the environment by lead occurs through deposit of airborne lead directly in water, and on streets and other paved areas from which some will be washed into waters. Airborne lead also is deposited in relatively high amounts on plants, along heavily travelled roads, and in lesser concentrations but over vast areas more distant from highways. Reduction of lead in gasoline will, therefore, result in reduced exposure of man, both directly from reduction in atmospheric lead, and indirectly from reduction of lead in dirt, dust, and at least to a minor extent, on and in foods.

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Office of Air Programs
5600 Fishers Lane, Rockville, Maryland 20852

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DEC 17 1971

EPA Strategy for regulating the use of lead as a fuel additive

The Administrator
THROUGH: Assistant Administrator for Air and Water Programs

1. Action required: A decision is to be made on EPA's strategy for regulating the use of lead as a fuel additive. EPA has indicated that an announcement on this matter would be made this month.

2. Statutory basis: Under the Clean Air Act, as amended, EPA can attack the lead problem via:

a. Federal regulation of the use of lead additives in gasoline under section 211 of the Act, based on the adverse effects of airborne lead on public health and welfare and/or the adverse effects of leaded gasoline on the performance of motor vehicle emission control systems.

b. Promulgation of national ambient air quality standards for lead under section 109 of the Act, which would mean that States would have to prepare and carry out implementation plans.

3. Regulatory objectives: There are two principal objectives:

a. To insure that public exposure to airborne lead is reduced to the degree which available scientific data indicates is adequate for protection of public health; analysis of the data suggests that attainment of an ambient air level of 2 micrograms (maximum 3-month average) would satisfy this criterion.

b. To insure that lead-free gasoline will be generally available for use in new automobiles, if an when they are equipped with lead-sensitive emission control systems (e.g., catalytic converters), and that lead-free fuel will, in fact, be used in such automobiles.

4. Strategy options: EPA can achieve the above objectives by way of the following alternative action plans:

a. Plan I: Promulgation of nationally applicable regulations under section 211(c)(1)(B) to provide for general availability of one grade of lead-free gasoline.

Lauerbach/Hanson:kma -12/17/71 - Official file located in Rm. 17B-22, Pkln. Bldg.

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b. Plan II: Promulgation of regulations under section 211(c)(1)(B), as in Plan I, plus promulgation of national ambient air quality standards for lead.

c. Plan III: Promulgation of nationally applicable regulations under sections 211(c)(1)(A) and/or (B) to provide for one grade of lead-free gasoline, if necessary for purposes of motor vehicle emission control, and/or to reduce the use of lead additives in regular and premium grades.

5. Plan I: Promulgation of nationally applicable regulations under section 211(c)(1)(B) to provide for general availability of one grade of lead-free gasoline by mid-1974.

a. Background:

(1) Plan I is based on the assumption that, beginning with the 1975 model year, new cars will be equipped with catalytic converters, the effectiveness of which would be impaired by the presence of lead in motor vehicle exhaust. There is no known, practical way of filtering the lead out of the exhaust stream before it enters the catalytic converter.

(2) If Plan I is implemented, use of lead additives would diminish, and ultimately cease, as new cars equipped with catalytic converters replace older cars using leaded fuel. The reduction in the use of lead additives would be approximately 10 percent per year. Thus, Plan I would also serve as a means of achieving EPA's health protection objective (assumed to be a 3-month average ambient air level of 2 micrograms), e.g., in regions where current lead levels are not above 2.5 micrograms, introduction and use of one grade of lead-free gasoline, beginning in mid-1974, would reduce the ambient air levels to 2 micrograms or less by mid-1976; in regions where current lead levels are highest (i.e., Los Angeles and Dallas, both in excess of 5 micrograms), Plan I, alone, would result in attainment of a 2-microgram level by mid-1981.

b. Pro:

(1) Among the various alternatives, Plan I would have the smallest economic impact on petroleum refiners and fuel consumers.

(2) Plan I provides not only for protection of emission control systems but also for protection of public health at no additional cost.

c. Con:

(1) There would be some places where an ambient air level of 2 micrograms would not be reached until mid-1981. Environmental groups will object strenuously.

(2) In the event that automobile manufacturers do not use catalytic converters, there would be no statutory basis for Plan I. Available data are inadequate to support a complete phase-out of lead additives in order to protect public health.

6. Plan II: Promulgation of nationally applicable regulations under section 211(c)(1)(B), as in Plan I, plus promulgation of national ambient air quality standards.

a. Background:

(1) As indicated above, introduction of one grade of lead-free gasoline in mid-1974 would result in attainment of an ambient air lead level of 2 micrograms by mid-1976 in regions where current levels are not more than 2.5 micrograms. There are, however, an estimated 22 regions where levels are higher. Promulgation of national ambient air standards by mid-1972 would trigger a timetable for their attainment in all places by mid-1976.

(2) Plan II would provide for national availability of one grade of lead-free gasoline plus State regulatory action to reduce the lead content of regular and premium gasolines in an estimated 22 regions.

b. Pro:

(1) A health-protection level would be reached in all places by mid-1976.

(2) The lead content of regular and premium gasolines would have to be reduced only in those regions with current lead levels above 2.5 micrograms.

(3) Assuming that there are just 22 regions with current lead levels above 2.5 micrograms. Plan II is the least-cost (in terms of total national costs) way of providing for attainment of a health-protection level throughout the Nation by mid-1976.

(4) Promulgation of a national ambient air standard insures that States will regulate not just the lead content of regular and premium gasolines, where necessary, but also (as part of their implementation plans) lead emissions from stationary sources in regions where such sources are responsible for ambient air lead levels in excess of the national standard.

(5) State regulations affecting regular and premium gasoline would vary among the 22 regions, but EPA has authority to impose restrictions on State implementation plans related to fuel additives, e.g., EPA is authorized to reject a State plan that provides for greater control of the use of fuel additives than is necessary to meet a national ambient air standard; it could also, through classification of regions, limit the variation in State regulations.

c. Con:

(1) Additional air sampling over the next twelve months could show that there are more than 22 regions with lead levels above 2.5 micrograms due to motor vehicle emissions.

(2) Even if States must regulate the lead content of regular and premium gasolines in just 22 regions, there will be varying regulations which, according to petroleum refiners, could complicate production and distribution of gasoline.

(3) Though EPA, as indicated above, has authority to reject State plans which provide for "too much" control of the use of fuel additives, exercising this authority would put EPA in a rather uncomfortable position.

(4) States already are burdened with the task of preparing and carrying out implementation plans for six pollutants. A substantial number of States will have real difficulty, particularly in carrying out their plans.

(5) Enforcement of State regulations affecting regular and premium gasolines will be difficult because most of the gasoline consumed in most States is produced outside the State; accordingly, States would have to rely largely on enforcement at retail outlets.

7. Plan III: Promulgation of nationally applicable regulations under sections 211(c)(1)(A) and/or (B) to provide for one grade of lead-free gasoline and/or to reduce the use of lead additives in regular and premium grades.

a. Background:

(1) Use of (A) and (B) would mean providing for national availability of one grade of lead-free gasoline for use in catalyst-equipped cars beginning in mid-1974 plus nationwide reduction of the lead content of regular and premium gasoline. The latter scheme would result in phased reduction to a maximum of 2.0 grams per gallon in premium and 0.8 grams per gallon in regular over a five-year period ending in 1977. This scheme, combined with the introduction of one grade of lead-free gasoline in mid-1974, would result in a 65 percent reduction in airborne lead levels by 1977, sufficient to insure

attainment of a 2-microgram level even in the places (i.e., Los Angeles, Dallas) where airborne levels currently are highest.

(2) Use of (A) alone would be necessary if catalytic converters, for any reason, are not used to meet EPA's motor vehicle emission standards, thereby making it impossible to mandate availability of one grade of lead-free fuel. Again, the regulations would provide for a phased, five-year reduction of the lead content of regular and premium, but the end-points would be lower, i.e., 1.6 grams in premium and 0.5 grams in regular.

b. Pro:

(1) Exclusive use of section 211 to regulate the use of lead additives would eliminate the uncertainty (and possibe varying regulations) associated with State implementation of national ambient air standards.

(2) If catalytic converters are not to be used, Plan III, relying on section 211(c)(1)(A) only, is far superior to Plan II. If one grade of lead-free gasoline is not introduced, then there would have to be widespread and widely varying State regulation affecting regular and premium gasoline in order to insure attainment of health-protection levels. There is reason to anticipate that automobile companies generally will maintain that catalytic converters cannot be ready in time for the 1975 model year; this would tend to raise substantial question as to the justification for, and timing of, an EPA regulation providing for availability of one grade of lead-free gasoline.

Ctrl—— (3) Uniform nationwide regulation eases the petroleum industry's production and distribution problems and government's surveillance and enforcement problems.

(4) The difficulty of minimizing contamination of lead-free fuel in a distribution system that also carries leaded regular and premium gasoline would be lessended as the lead content of all regular and premium gasolines is reduced. — Ctrl

c. Con:

(1) Plan III involves reduction of the lead content of regular and premium fuels in all places, even those places where such reduction is not really necessary for health-protection.

(2) Since Plan III would affect all U.S. gasoline, it would be more costly than Plan II. To determine the exact difference would

require extensive economic analysis. An estimate is that it would amount to $500 million more (for a five-year phased reduction) than Plan II. On the average, the additional cost to consumers would be less than one cent per gallon.

(3) Lead emissions from stationary sources would have to be attacked separately under other provisions of the Clean Air Act. In all likelihood, new source performance standards would be established under section 111. States, in turn, would be obliged to establish emission standards for existing sources.

8. Other considerations:

a. Effect on small refiners: Any regulatory action impinging on the use of lead additives could have serious consequences for small refiners. The slower the rate of reduction, the more gradual will be the impact on small operators. But if there is a transition to lead-free gasoline because of catalytic converters, then most small refineries may well have to stop producing gasoline. It should be noted, however, that there is a long-standing trend in this direction.

b. Aromatics: If lead additives are not used, other octane boosters must be added. Generally, increased amounts of aromatic hydrocarbons would be added. Lead-free fuel would be used by new cars, which, beginning with the 1975 model year, will have to be equipped to reduce hydrocarbons, including aromatics, by 90 percent from current levels; accordingly, any increase in aromatic content of gasoline will have a negligible effect on aromatics in exhaust gas. Low-lead fuel would be used by pre-1975 cars, but such fuels can be made without substantial increases in aromatic content. In short, none of the Plans outlined in this paper would result in a worsening of photochemical smog problems. It should be noted, however, that the extent of increased use of aromatics depends on how quickly refiners must reduce the use of lead additives. Any regulatory scheme providing for a more rapid rate of reduction during the next five years than is required by Plan III could increase the photochemical reactivity of automobile exhaust emissions. These conclusions about aromatics in relation to smog are also applicable to exhaust emissions of cancer-producing polynuclear aromatics (PNA).

9. Conclusions: Based on the above considerations, it is concluded that Plan III is the best choice. It is the only practical means of providing for public health protection by mid-1977 regardless of whether catalytic converters requiring lead-free fuel are used for motor vehicle emission control. If they are not, the end-point of the lead-reduction schedule

can be modified to insure attainment of a health-protection level in all places by mid-1977. If catalytic converters are not used, Plan II would result in an unacceptable degree of variation in State regulations, while Plan I would have no statutory basis.

10. Recommendations:

a. It is recommended that you adopt Plan III as EPA's strategy, subject to your approval of the necessary proposed regulations, which should be ready for in-house EPA review within the next several days.

b. The Act requires public hearings on proposed regulations only if they are based on emission control system requirements and only if such hearings are requested. Nevertheless, because of the significant and controversial nature of the lead question, it is recommended that you announce your intention to have hearings in any event.

/s/ Irwin Auerbach

for John T. Middleton
Deputy Assistant Administrator
for Air Programs

D

Natural Resources Defense Council, Inc.

15 WEST 44TH STREET
NEW YORK, N.Y. 10036
212 869-0150

A 254

Washington Office
917 15TH STREET, N.W.
WASHINGTON, D.C. 20036
202 737-5000

Western Office
664 HAMILTON AVENUE
PALO ALTO, CALIF. 94301
415 327-1080

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Executive Director

October 2, 1974

BY CERTIFIED MAIL

Russell Train, Administrator
U.S. Environmental Protection Agency
Washington, D.C. 20460

Dear Mr. Train:

Section 108(a)(1) of the Clean Air Act requires the Administrator, within 30 days after enactment of the Clean Air Act, to list lead. The Administrator has not done so. On many previous occasions, including a petition of March 19, 1973, and supplemental petition of May 15, 1973, and in Petitioners' Brief and Reply Brief in NRDC v. EPA (D.C. Cir., No. 74-1023), we have explained why the legislation demands such action, asked for such action to be taken, and notified EPA of its failure to comply with the law.

I am calling this obvious failing to EPA's attention once again. It has become clear, particularly in recent months, that EPA's failure to list lead is part of a systematic violation of the Clean Air Act. Congress enacted section 108 for the purpose of putting the protection of public health on a timetable; section 108(a)(1) triggers the timetable of sections 108-110. But, since enactment, only one substance, nitrogen oxides, has been listed under section 108. In contrast, Administration officials promised both the House and Senate Committees* that certain substances, including lead, would be treated as criteria pollutants. Moreover, EPA itself, in the months following enactment, had plans to list numerous substances in keeping with the mandatory duty of section 108(a)(1). The policy of side-stepping the mandate of section 108(a)(1), apparently formulated and promoted by EPA's Office of Air Quality Planning and Standards,

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* Hearings on S. 3229, 3466, 3546 Before the Subcomm. on Air and Water Pollution of the Sen. Comm. on Public Works, 91st Cong., 2d Sess. 196 (1970); Hearings on HR. 12934, 14960, 15137, 15192, 15848, and 15847 Before the Subcomm. on Public Health & Welfare of the House Commerce Comm., 91st Cong, 1st & 2d Sess's. 293 (1970); S.REP. No. 91-1196 91st Cong., 2d Sess. 9 (1970).

[continued]

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effectively guts that part of the legislation designed to protect health from many pollutants on a timely basis. EPA's Air Quality Criteria Advisory Committee has begun to balk at the farce of no air quality criteria being issued since the first days of the Act.*

I would be pleased to discuss this general problem, if you think it would be useful. But, I do intend to file an action in District Court on the lead aspect of the problem shortly since EPA's failure to list lead will, in all likelihood, cause a tripling of lead in gasoline in New York on January 1, 1975. Accordingly, this letter is being sent pursuant to 40 C.F.R. §54. But, given our previous actions, such notice is unnecessary since notice has already been given and received of all matters required by §54.3.

Sincerely,

David Schoenbrod

S/t

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* The following is an excerpt from the June 27, 1974, meeting of the National Air Quality Criteria Advisory Committee, Summary and Follow-up Report, p. 15: "The discussion of air quality criteria for lead broadened into a more general expression of concern regarding air quality criteria for pollutants other than the six for which they exist and regarding EPA's philosophy with respect to additional air quality criteria and standards. There was reference to a listing of Reports on Pollutants, included in members' folders, in which the Committee has been involved and it was noted that, with the exception of the Revised Chapter 5 of the Air Quality Criteria for Sulfur Oxides which came out in 1973, no criteria document has been issued since 1971. It was also noted that the reports on PPOM, Suspended Sulfates, and Sulfuric Acid, Aerosols, Cadmium, Polychlorinated Biphenyls, Manganese, and Vanadium on which the Committee has been consulted, have not yet been published and, insofar as was known to the Committee, had not resulted in any control actions. Concern was expressed about the purposes and the usefulness of these reports and about the Agency's apparent reluctance to issue or develop air quality criteria and an apparent preference for new source performance standards." Attached hereto is an excerpt from the same Committee meeting as to how this problem affects the control of lead pollution. pp. 13-14, "Summary and Follow-up Report."

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As already indicated, it was pointed out that the problem of airborne lead from stationary sources should not be approached as a basically local problem with the implication that there is no need for national ambient air quality criteria and standards.

It was pointed out that, as stated in the Introduction (p. 11), any one source or group of sources must be considered in relation to the contribution made by other sources.

It was pointed out that airborne lead is ubiquitous, that its presence in the ambient air results from numerous and diverse sources and that it appeared, therefore, to meet the requirements for issuance, at the appropriate time, of air quality criteria for the purpose of establishing national primary and secondary ambient air quality standards.

It was pointed out that air quality criteria and standards are needed as reference points for judging the adequacy of current emissions control technology.

It was pointed out that the problem of lead from stationary sources was one of existing rather than of new sources and that the EPA regulations, required under section 111(d) of the Clean Air Act, as amended, which would establish a procedure for each State to control existing stationary sources of airborne lead had not yet been issued.

It was pointed out that air quality criteria and standards would provide a basis for the relocation, away from residential areas, of certain stationary sources which apply current control technology but continue to produce undesirable overexposure of humans.

There was agreement, in essence, with Recommendations No. 4 to 8 regarding needed studies and more and better information.

There was great concern about the likelihood, indicated in response to questions, that there will be no further EPA funding of research on the health effects of airborne lead. Pointing to the controversial nature of the health implications of airborne lead and the recognized uncertainties and gaps of knowledge, a majority of members present went on record wishing to express their concern to the Administrator and stressing the need for continuing desirable and effective research in this area.

pp. 13-14, "Summary and Follow-up Report," June 27, 1974, meeting of the National Air Quality Criteria Advisory Committee

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UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

OFFICE OF THE
ADMINISTRATOR

Dear Mr. Schoenbrod:

This is in response to your letter of October 2, 1974, urging the Environmental Protection Agency to list lead as a criteria pollutant under Section 108 (a)(1) of the Clean Air Act and advising EPA of your intention to file a citizen suit under Section 304(a)(2) of the Act, alleging that the Administrator has failed to perform a non-discretionary duty by his decision not to list lead and to issue air quality criteria and national ambient air quality standards for this pollutant.

I understand that your suit was filed in Federal District Court for the Southern District of New York on October 21, 1974. This action followed an order of the U.S. Court of Appeals for the District of Columbia Circuit dismissing your petition for review of EPA's regulations controlling lead additives in gasoline for want of jurisdiction. NRDC v. EPA, No. 74-1023 (D.C. Cir., September 11, 1974). The grounds of your suit under Section 304 of the Clean Air Act are substantially the same as the grounds for your earlier petition for review of EPA's regulations controlling lead additives in gasoline. Under these unusual circumstances, EPA will not object to your failure to provide sixty days notice of your intention to file a citizen suit as required by Section 304(b)(2).

EPA's position on the legal issue you raise has been set forth before, and I have only summarized it in this reply. I would like to respond in greater detail to your criticism of EPA's choice of regulatory strategies for lead and other pollutants under the Clean Air Act. Perhaps an explanation of the considerations we take into account in developing regulatory programs will promote better understanding of our policies.

A. Action to List Lead Under Section 108 Is Discretionary.

As you know, it is EPA's judgment that the decision whether to list lead as a criteria pollutant under Section 108 of the Clean Air Act is discretionary with the Administrator. (39 F.R. 33734, 33740, December 6, 1973.) Section 108(a)(1) of the Act states that the Administrator shall, within 30 days after the date of enactment of the Clean Air Amendments of 1970, publish, and from time to time revise, a list which includes each air pollutant --

(A) which in his judgment has an adverse effect on public health and welfare;

(B) the presence of which in the ambient air results from numerous or diverse mobile or stationary sources; and

(C) for which air quality criteria had not been issued before the date of enactment of the Clean Air Act Amendments of 1970, but for which he plans to issue air quality criteria under this section. [Emphasis added.]

In our opinion, conditioning the listing of pollutants under Section 108 upon the Administrator's judgment as to health effects and his "plans" to issue criteria falls far short of a mandate to list any particular pollutant.

The reference to lead and other pollutants as candidates for the issuance of criteria in the Report of the Senate Public Works Committee, upon which you rely, has never been regarded by EPA or by the Committee as a legal requirement to list lead or any other substance as a criteria pollutant. Officials of the Department of Health, Education, and Welfare, EPA's predecessor in regulation of air pollution, informed the Committee considering amendments to the Clean Air Act that current planning called for the issuance of criteria for twenty-three different pollutants. Under the circumstances, it is not surprising that HEW's planning contemplated criteria for practically every known pollutant. The issuance of criteria was the principal basis for any regulatory action under existing legislation, a condition which was changed by Congress in amending the Clean Air Act.

The Amendments to the Clean Air Act ultimately enacted in 1970, provided a variety of new tools for the abatement of pollution, such as the authority to set Federal standards for motor vehicle fuels. We believe the Agency has broad discretion in selecting the most effective strategies authorized by the different provisions of the Act. In addition, the Congressional Committees overseeing EPA's activities have not criticized EPA's choice of criteria pollutants, much less protested that the listing of certain substances is mandatory. Under these circumstances, EPA adheres to its consistent interpretation that Section 108 does not impose a non-discretionary duty to list lead or any other substance as a criteria pollutant.

B. Reasons for the Decision to Regulate Automotive Lead Emissions Under Section 211 of the Act.

In 1971, EPA was required to take steps to assure the general availability of unleaded gasoline for use in catalyst-equipped vehicles expected to be marketed in 1974. Many auto manufacturers have chosen catalyst emission control systems to meet the auto emission standards, and catalysts are deactivated by leaded gasoline. Since the general availability of unleaded gasoline meeting national standards was to be required by regulation, the question EPA addressed was how to go about reducing lead levels in leaded gasoline to reduce total automotive lead emissions to a level that would protect health, considering the reductions that would also be occurring due to the phase-in of unleaded fuel. The alternatives considered were to establish national fuel standards for leaded gasoline under Section 211 or to set an ambient air quality standard for lead that would require the States to regulate lead in gasoline to whatever extent was necessary to make up the difference between the ambient standard for lead and the levels that would be reached solely by the Federal regulations phasing in unleaded gasoline.

The listing of lead as a criteria pollutant under Section 108 of the Clean Air Act triggers the issuance of air quality criteria and a national ambient air quality standard under Section 109, to be implemented by the States under Section 110 of the Act. The air quality standards themselves -- for example, the sulfur or carbon monoxide standards -- are established by determining the level of the pollutant in the air that corresponds to evidence of adverse health effects (a dose-response relationship) and then reducing that level to the degree

necessary to provide a margin of safety. The result is a numerical standard considered sufficient to protect public health. The States are required by Section 110 to control emissions of pollutants subject to the national ambient air quality standards to the extent necessary to assure compliance with the standards in all areas of the State. Therefore, State controls can be expected to vary in accordance with the severity of the particular pollution problem in the State.

The Act establishes specific timetables for the attainment of the primary ambient standards to protect public health. In addition, the cost and present technological feasibility of meeting ambient standards necessary to protect health are not to be taken into account in setting the standards. Thus, the issuance of a national ambient air quality standard requires definition of a numerical standard which the States must implement on a specific timetable, without taking into account cost and technical feasibility.

As we explained in Respondent's Brief at 15-16, NRDC v. EPA, supra, the decision to regulate automotive lead emissions under Section 211 of the Act was made on the grounds that (a) uniform Federal standards would make industry compliance simpler than a proliferation of differing State standards, (b) Federal enforcement directed at the comparatively small number of refiners rather than State enforcement at the distributor-retailer level would be more efficient and effective, and (c) the States were hard pressed to implement the six ambient air quality standards already promulgated and should not be required to undertake another enforcement task when equally effective alternatives were available.

Two factors did not enter into the decision to act under Section 211: the feasibility of setting an ambient standard for lead and the existence of statutory timetables for action under Section 108-110. The Agency did not realize then that it would be difficult, if not impossible, to establish a defensible numerical value for an ambient air quality standard for lead; and there was, at the outset, no substantial difference between the projected timetable for achieving reductions in lead emissions under Section 110 and 211. Both of these conditions changed. The original scientific basis for a specific ambient air quality goal was subsequently discredited, and the final attainment date of the complete lead reduction program was delayed by two years due to the time required to reevaluate the evidence on the health effects of lead.

C. Present Feasibility of Establishing a National Ambient Air Quality Standard for Lead.

When the Federal lead regulations controlling lead in leaded gasoline to protect health were proposed in February, 1972, EPA believed that the attainment of ambient lead levels of 2 micrograms per cubic meter of air (2 ug/m^3) should be the goal of the lead reduction program. This number would have been proposed as the national ambient air quality standard, had EPA opted for that strategy. The lead regulations proposed and promulgated require a 60-65% reduction in lead in gasoline and will achieve that goal, but the 2 microgram level is no longer considered to be a meaningful measure of the risk of adverse health effects from lead emissions.

The 2 microgram goal was based on the Goldsmith-Hexter equation, developed in California, which attempted to relate lead concentrations in the blood of various groups of men with different atmospheric lead exposures. EPA concluded from this analysis that elevated blood lead levels and excess body burdens were associated with airborne lead levels exceeding 2 ug/m^3 as a quarterly average.

In the course of the comments, considerable doubt was cast on the validity of this equation in establishing a dose-response relationship as a basis for a 2 microgram goal for reduction in lead emissions. The evidence needed to correlate airborne lead levels with human blood levels indicating excessive exposure and to establish a dose-response for airborne lead and adverse health effects was found to be lacking because other sources of human lead exposure tend to mask the effects of human exposure to airborne lead. The primary source of lead exposure for most persons is the diet; some lead occurs naturally in food and water. And for the major target of EPA's lead control program, urban children, ingestion of lead from lead-contaminated dustfall and leaded paint in their environments is another important source of exposure to lead.

Because of these other sources, it has not yet been possible to quantify the contribution of airborne lead and dustfall lead derived from the air to high blood lead levels in adults and children. For these reasons, I concluded that "it was difficult, if not impossible, to establish a precise level of airborne lead as an acceptable basis for a control strategy." 39 F.R. 33734 (December 6, 1973). Such a precise level

need not be identified to conclude that lead emissions from motor vehicles constitute an endangerment of health under Section 211 of the Act, but the identification of such a level is necessary to issue a national ambient air quality standard for lead.

Notwithstanding the gaps in the information needed to establish an ambient standard, I am convinced that lead emissions present a significant risk to urban populations and that the risk must be reduced to the extent feasible. EPA's lead regulations for gasoline are the result of that assessment, and we believe that Section 211 provides a firm basis for taking reasonable steps in the face of an unquantified and perhaps unquantifiable endangerment to health. The Agency is also committed to considering whether further controls on lead in gasoline are warranted as new information develops. See 39 F.R. 33734, 33740.

D. EPA Policy and Procedures in Selection of Pollution Control Strategies.

Congress has given EPA a variety of tools for control of air pollution. In addition to the authority to set national ambient air quality standards under Section 110, the Clean Air Act provides authority to establish emission standards for new sources (§ 111), hazardous pollutants (§ 112), new motor vehicles (§ 202), motor vehicle fuels and fuel additives (§ 211), and aircraft (§ 231). The choice of the optimum control strategy for a pollutant is one of the most important regulatory decisions we make.

The procedure followed by EPA staff in identifying the most appropriate control strategy is called a Preferred Standards Path Analysis. Factors to be taken into account include the nature and severity of the health effects of the pollutant; the type and dispersion of the emissions of the pollutant and its sources; the extent to which the sources are controlled by existing regulations at the Federal, State, or local level; and the choice of legal authority and enforcement mechanisms available under different provisions of the Act. With the results of this analysis, we decide what strategy should be adopted.

In developing a regulatory strategy for lead from stationary sources of lead, for example, EPA has reviewed the health effects of lead emissions from stationary sources such as smelters, the adequacy of the data to support an ambient standard, whether lead emissions from these sources tend to be localized or widely dispersed and the extent to which lead particulate emissions are controlled by existing particulate control or sulfur oxide control techniques which require the removal of particulates as part of the process of sulfur control. By this process, EPA is identifying sources of lead emissions not adequately controlled by existing or proposed standards and is proceeding to establish lead standards for those classes of stationary sources.

EPA's approach to the analysis of control strategies under the Clean Air Act was reviewed by Dr. B. J. Steigerwald, Director of the Office of Air Quality Planning and Standards, at the September 12, 1974, meeting of the National Air Quality Criteria Advisory Committee. I am sure that Dr. Steigerwald and other EPA officials with responsibility for recommending regulatory programs for lead and other pollutants would be glad to discuss our policies with you at any time.

In summary, we recognize that NRDC believes that EPA's lead regulations for gasoline are not sufficiently stringent and believes that the establishment of a national ambient air quality standard for lead would cure what you view as deficiencies in EPA's lead control program. We remain convinced, however, that the lead control strategy being implemented by the Agency is most firmly based on other sources of authority in the Clean Air Act and that this strategy affords the most effective means of achieving the reduction in lead emissions we both desire.

The remedy NRDC seeks -- a determination that EPA must choose ambient standards as a control strategy for lead and many other pollutants mentioned in the

legislative history -- would severely impede EPA's efforts to establish sound public health priorities and to develop regulatory programs best tailored to the pollutant sought to be controlled. We are, therefore, unable to concur in your interpretation of our authority under the Act.

Sincerely yours,

Russell E. Train

David Schoenbrod, Esq.
Natural Resources Defense Council
15 West 44th Street
New York, N.Y. 10036

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74-AP-19-PNWIS MEETING
BOISE, IDAHO
17-19 NOVEMBER 1974

PROCEDURES EMPLOYED FOR A STUDY OF LEAD IN DUST, SOIL AND THE AMBIENT AIR

A. J. YANKEL AND I. VONLINDERN

IDAHO DEPARTMENT OF HEALTH AND WELFARE

Exhibit A

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ABSTRACT

Idaho's Silver Valley Region Consists of a narrow valley approximately two miles wide and 30 miles long. The valley has been the center of extensive mining and related operations for over 100 years. Presently, there are 14 active mines in this area, as well as a large lead/zinc smelting complex that has been in existence for over 50 years.

Since mid-1970, the Department of Health and Welfare has been monitoring the levels of lead in the ambient air in the vicinity of Kellogg, Idaho. In 1973, the levels of lead began to increase. Although no specific numerical air quality standards exist for lead and no health problems attributable to the lead concentrations had been reported, the Department concluded that a special study of the situation was warranted. Shortly after a limited environmental study was initiated by the Department, in early 1974, two cases of lead absorption in children from Kellogg was reported. In response, the environmental survey was intensified and carried out in conjunction with a comprehensive study of human lead absorption.

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INTRODUCTION

Following the investigation of two cases of lead absorption in children, the Idaho Department of Health and Welfare determined that the planned study should be revised and expanded. At that time, the Department had only limited information available concerning environmental contamination in the area. Three hi-volume air particulate samplers had been operating in the Kellogg area since 1971. These samples had been analyzed for lead content. Approximately 30 soil and vegetation samples had been collected and analyzed for lead through 1973. An examination of the existing literature was found to be largely inconclusive. It was not clear at what levels ambient air concentrations or lead levels in the soil or dust became hazardous. However, in several instances, concentrations much lower than those suggested in Kellogg were cited as being potentially hazardous.

A National Academy of Sciences publication[4] refers to the swallowing of lead-contaminated dusts (as low as 1500 ppm) as possibly accounting, in a large part, for the higher mean blood lead content in urban children. The Environmental Protection Agency[3] suggests in its position paper on the health effects of airborne lead that street dusts with mean concentrations of 4000-5000 ppm could be significant contributors to excess lead absorption. Vostol, et.al.[6] in reference to Needleman and Scanlon[5] alludes to the theory that unintentional ingestion of household dusts with concentrations of 1000-2000 ppm can exceed the daily permissable intake of lead in children. EPA in reference to Baltrap[1] cite lead contamination in dirt as having one half the potential hazard that lead in paint has in this country.

Airborne lead levels in urban Los Angeles have long been source of serious concern. The National Academy of Sciences[4] cites a yearly average of 2.5 ug/m^3 and a monthly averages as low as 6.4 ug/m^3 as warranting concern.

Carnow[2], et al, referred to lead levels of less than 5 ug/m^3 as contributing significantly to excess lead absorption near a smelter in El Paso, Texas. The Environmental Protection Agency[3] believes that concentrations even less than 2 ug/m^3 can effect blood lead levels.

Soil and dust samples obtained from the Kellogg area in early 1974 had lead values ranging from 5000-180,000 ppm. Monthly average lead concentrations in the ambient air were consistently over 10 ug/m^3. Monthly averages in Smelterville had been greater than 30 ug/m^3 at a distance of nearly one mile from the smelter.

Moreover, the ambient air quality in relation to lead had been steadily deteriorating since the beginning of records in 1971. A regular increase in ambient air lead levels had been evident. This increase can be seen in Figure 1 (lower plot). The upper plot in Figure 1 represents readings obtained for total suspended particulate. As the ambient lead and particulate concentrations do not increase proportionately, an increase in percent lead in the particulate matter has been evident. This trend is shown in Figure 2.

Because these are levels of concern and because the situation seemed to be deteriorating, it was imperative that the sources of this contamination be identified and quantified as quickly as possible. These observed percentages of lead in the particulate matter are much higher than any observed in the soil thus far. This would suggest that the bulk of the lead in the ambient air originates from sources of high lead concentration. Those sources would be concentrating and smelting activities.

Although most of the literature deals with lead contamination from automotive or paint sources and the propriety of extrapolating, such data to lead contamination from smelting and mining activities is unclear. It was easily concluded that the potential for an extreme health hazard existed in the Kellogg area and a comprehensive and extensive study was initiated.

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DESIGN OF STUDY

Among the many necessary determinations to be made in this study were: 1) the extent of heavy metal contamination through both the elements of the environment and the entire mining district, 2) the identification and assessment of the magnitude and impact of both active and inactive sources of contamination, 3) the mechanisms by which humans acquire abnormal levels of heavy metals intoxication, 4) the specific toxicity and mobility of the contaminants in the various environs, and, 5) the means by which the environment could be modified to eliminate the hazards.

So that these determinations could be made as quickly and as comprehensively as possible with what resources were available, an ordered and organized study plan was developed. It was decided to collect and analyze data in three separate phases. Each phase was to have its own objective and mode of study and yet be easily integrated into the overall study. The three phases are outlined below.

PHASE I

The purpose of Phase I was to collect general, but comprehensive, environmental data as quickly as was possible. The objective of collecting and analyzing this data was to identify the extent of contamination through the entire valley and, as possible, the sources of contamination. This information would then be used to confirm the extent and level of contamination and aid in defining priority study areas in later phases.

PHASE II

This phase was designed as an intenseive study of the elements of the environment throughout the valley. The purpose was to identify the intensity and range of contamination and to obtain a quantitative estimate of the contributing sources. Also, specific information on the chemical and physical

properties of the pollutant was sought in order to determine its mobility, transport mechanisms, particle size, solubility, toxicity and potential environmental impact. This study was also designed to provide a comprehensive and accessable data base for future environmental and health related studies that would address the valley and possible environmental measures that would improve the situation.

PHASE III

This phase was designed as a specific survey of home environments in conjunction with a blood lead survey of children in the valley. The primary purpose was to correlate blood lead levels with medical histories as well as housing parameters and lead contamination in the immediate environment of the children. It is hoped that the information gathered in this phase will identify those factors in the immediate environment that contribute to excess lead absorption.

METHODS

PHASE I

Historically, ambient air concentrations for the three monitors in Kellogg and Smelterville were well above the level of concern. It was necessary to expand the sampling network throughout the valley so that the scope and extent of elevated ambient air concentrations could be determined. Twenty-four hour samples were collected every other day at each of nine sampling stations in order that reasonably accurate monthly averages and variations could be calculated. The stations are listed in Table I. The three previously existing monitors are indicated by a star. The additional stations were installed in late April and early May of 1974.

TABLE I

STATION NAME	LOCATION	DISTANCE FROM SMELTER	DIRECTION FROM SMELTER
Cataldo	Commercial Building, Cataldo	7.9 mi.	West
Pinehurst	Elementary School, Pinehurst	3.5 mi.	West
*Smelterville	City Hall, Smelterville	.9 mi.	West
Silver King	Silver King School, Smelterville	.3 mi.	West
*Kellogg Medical Center	Kellogg Doctors Clinic, Kellogg	1.8 mi.	East
*Kellogg	City Hall, Kellogg	2.0 mi.	East
Osburn	Radio Station, Osburn	7.9 mi.	East
Wallace	Post Office, Wallace	11.8 mi.	East
Mullan	Elementary School, Mullan	18.5 mi.	East

During the first ten days of June the Phase I soil and vegetation samples for this phase were collected from general sites (schoolyards, parks, graveyards, etc.) within each of the population centers in the valley. The sites were selected on the basis of reasonably homogeneous vegetation and land use. A record was made of the location, date, present land use, distance and direction to the nearest road, percent vegetation cover and the type of vegetation. From each site three soil samples and two samples of each type of vegetation were taken.

The soil samples were collected by visually dividing the site into five equal areas. Within each area, one randomly selected shovel full of the top 3/4 inch of soil was taken. Vegetation and organic material was removed as necessary. All soil was placed in a bucket and thoroughly mixed. This composite was then passed through a 1.7mm mesh screen to remove the large particles and organic matter. The screened material was once again thoroughly mixed. From this composite three smaller samples were selected at random for analyses.

Vegetations for Phase I were collected at, or near, the point that each soil sample was collected. Each sub-sample was obtained by cutting off the vegetation within two inches of the ground. Care was taken never to mix species in any samples and a grass sample was always obtained where possible. Each

sub-sample was placed in the same plastic bag. Care was taken not to disturb any particulate matter on the vegetation. When all five sub-samples were in the bag, the material was mixed and two samples were withdrawn for analysis. Leaf samples were taken by sampling leaves from several branches on a tree. Leaves were cut at the base of the petiole and placed in bags, mixed, and split. One of each pair of vegetation samples was washed before analysis. The other was not washed.

PHASE II

Phase II is the intensive environmental survey of the Silver Valley. A sampling grid was designed using the main stack of the smelter as a focal point and constructing equi-distant arcs drawn at logarithmic distances from the stack. Four sample sites were located on each arc where it intersected the river. Two sample sites were selected on the valley floor, one on each side of the river. The other sites were located on the arc at an elevation 75 feet above the river, one on each side of the valley. Seven samples were collected at each sampling site: grass, all other vegetation, litter (organic matter currently being incorporated into the soil), soil at a depth of 0-½", soil ½-1½", soil 1½-3", and soil 3-6". Phase II is designed to show the amount of lead that has been deposited as a result of fallout versus that which is found on the valley floor which is also influenced by mining activity or flood deposition. It is hoped that some indications of the mobility and residence time of lead in the environment can be obtained from this information.

Approximately 20 variables have been recorded for each sampling site and will be used in the analysis of Phase II data. Some of these variables are as follows: distance and direction from smelter, distance and direction from river, elevation above river, type of topography, aspect of hillside sites, soil type, amount of visible erosion, percent of vegetation cover, slope of

hillside sites, vegetation type present, present land use, distance and direction from nearest road.

Chemical and physical data on the air pollutant were also collected in this phase. Particle size distribution in the ambient air was obtained using both cascade impactors and Anderson Head impactor units. Percent lead oxides in particulate matter were obtained using an acetate leaching process. Through contracts initiated on behalf of the Department by the Environmental Protection Agency, an extensive network of hi-volume monitors and cascade impactors is currently in operation in the valley. This special ninety day study is to provide very specific and detailed information on air contamination in the immediate Kellogg-Smelterville area.

PHASE III

For the purposes of this phase of the study, seven general areas were set up (partly on the basis of Phase I results) to more conveniently group the samples that were taken with respect to anticipated lead levels. Area I consisted of all homes within one mile of the smelter, Area II - 40% of the homes within 1-2½ miles from the smelter, Area III - 33% of the homes within 2½-6 miles from the smelter, Area IV - 33% of the homes within 6-15 miles, Area V - 33% of the homes within 15-20 miles, and Areas VI and VII (100% of the homes) were control areas. These areas and the percentage of homes to be surveyed in each area were determined necessary to insure statistical significance.

In each area, a team consisting of one pediatrician, one public health nurse, and two environmentalists went door to door on the basis pre-determined by the statisticians. For example, in Area III where 33% of the homes were to be surveyed, the team would begin at one home on a particular street and then skip the next two homes stopping again at the third home. This pattern would be

followed throughout Area III. If there were no children at a particular house, or if the parents did not consent to having their children's blood drawn, the team would record this and move on to the next pre-determined house; in this case the third house down the street. If no one was home at a particular house, the address would be recorded and the team would return at a later time. If there were children at a pre-designated house, then the nurse and pediatrician would obtain written parental permission, a blood sample from each child between 1 and 9 years old, and a medical history.

The environmentalists sampled soil, vegetation, dust and paint at each home where a blood sample was obtained. Inside house dust was gathered from a vacuum cleaner in the home if that vacuum cleaner was only used to clean the inside of that home. If a vacuum cleaner was not available, the environmentalist used a modified hi-volume stack sampler to gather house dust on a pre-weighed filter. In some cases where an outside porch was present, an outside dust sample would also be gathered with the use of the hi-volume stack sampler. Soil and vegetation were sampled in the same manner as Phase I. If there were two distinctly different areas of the yard or if there was a play box, separate samples were gathered from each area. If a garden was present, samples of lettuce, beets and carrots were obtained. Chips of paint were taken from both inside and outside of the home. Tap water samples were obtained from several of the homes at a later date.

The dust, soil, water and vegetation are being analyzed for Pb, as well as Zn, Cd, As, Hg, and other heavy metals. The pH of the soil is also being determined and the vegetation is being analyzed as washed and unwashed. Only Pb is being analyzed in the paint samples.

DISCUSSION OF RESULTS

At the time of this report, only results from Phase I are available. A portion of Phase III results are complete but no attempt at analysis will be

made until all the information is available. Phase II samples were assigned a lower laboratory priority than those in Phase III. For this reason, and because the complexity of Phase II requires machine analysis, results from this phase will not be available.

The purpose of Phase I was to find where lead was present in the general environment and what possible sources of lead contamination have contributed, or continue to contribute, to the problem. It must be remembered that in this phase, the specific home environment is not addressed, but the study was designed to observe the general environment, (air, water, soil and vegetation). The results for each element of the environment follow.

AIR

The sources of air contamination by lead identified thus far in the valley are as follows:

TABLE II

SUSPECTED SOURCE	CONCENTRATION OF Pb	VOLUME	EFFECT AREA
Area Sources			
Automobiles	100 ug/m^3 in exhaust	~1T/mo	~10^2 ft. roads
Soil & dust	0-2%	?	Entire valley
Ore & concentrate transportation	5-60%	?	~10^2 ft. railroads
Point Sources			
Bunker Hill Smelter Blast Furnace & Main Stack	50-70%	~20-100T/mo	Entire valley
Bunker Hill Fugitive Sources	20-70%	~10-20T/mo	~10 miles
Milling Operations	5-15%	?	~1 mile

Currently the Department, through the services of EPA contractors, is having an intensive study of both point and fugitive emissions done. This study should quantify all sources and provide more accurate information for the above table.

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At present, three months of ambient air data have been obtained from the 9 hi-volume sampler network. Analysis of each of these months shows the same general pattern. Levels decrease regularly with distance from the reference point. If the source of an airborne contaminant is a point source or a confined area source in relation to the dimensions of the sampling network, then the dilution pattern should be logarithmic with respect to that source. In other words, a plot of concentrations versus the logarithm of distance from the virtual source should be linear. This criteria is fulfilled for these particular months as can be observed in Figures 3, 4 and 5. On this basis, it would seem that the primary source of airborne contamination during these particular months originated in the area of the Bunker Hill Complex.

SOILS

Significant and abnormal levels of lead were found throughout the valley. Levels of lead higher than those found in the existing literature are consistently observed in the Kellogg-Smelterville area. Several of the samples were screened for other heavy metals (zinc, cadmium, mercury and antimony), see Table III. It is suspected that the cadmium levels observed may constitute a human health hazard in some areas. Antimony and zinc are present in significant amounts in many areas; however, the health effects of the environmental contamination of these metals is not known. A local veterinarian has cited many animal deaths in the area as caused by antimony and cadmium, as well as lead poisoning. Because of the immediacy of the problem, attention was focused on lead and all analyses refer to lead levels. In comparison to published information, the lead levels in the communities of Kellogg and Smelterville are as high, or higher, than any recorded elsewhere.

Several sources of soil contamination of significant magnitude were identified throughout the valley. They include deposition of air and water-borne contaminants and the physical incorporation of solid dumped contaminants. A listing of possible sources follows:

Air Born Sources
- -dust blowing from other areas
- -deposition of particulate matter

Water Born Sources
- -deposition from polluted waters
- -deposition of flood residue
- -transport by surface runoff

Contamination By Solids
- -mine tailings disposal
- -jig tailings disposal
- -low grade ore disposal
- -ore and concentrate spills
- -tracking of material by transportation vehicles

Because of this variety of sources, the level of observed lead concentrations in the soils of the valley is somewhat erratic. There are several small isolated areas throughout the valley that are observed as having high levels. Usually those high levels can be attributed to one or more of the above. Areas around old and present mills have high concentrations. Tailings disposal sites are high in lead and other metals. Flood plain sites show increased lead levels. Several other examples are common.

However, it is obvious that high levels are continually observed in the Kellogg-Smelterville area. These concentrations seem to be centered at the Bunker Hill Complex. This is not surprising as this area is the focal point of the lead industry in the valley. Certainly some of this soil contamination is a result of discharges and effluents from the smelter complex. Others are a result of ores and concentrates moving toward the smelter. Disposal of tailings and the use of mine wastes for fill, etc., contributes

to this concentration. Essentially this area is, and has been, for over 80 years, the center of a large, active, and everchanging industry. The lead contamination in the soil of this area represents these many years of activity.

It should be remembered that Phase I was an intensive study designed to determine what degree of contamination is present throughout the valley. The values shown in Table I were not taken in conjunction with the blood survey and thus cannot be directly or indirectly related to health problems. The results of Phase I soil samples are shown in Figure 6. The results have been averaged for each town and one standard deviation is plotted. It can be seen that the same general pattern exists as is shown with the ambient air samples with higher concentrations around the smelter complex and decreasing concentrations with distance from the smelter.

VEGETATION

The lead levels observed in vegetation are a product of both physiological uptake from contaminated soils and the absorption and deposition of particulate matter from the atmosphere. We would, therefore, expect that the vegetation would reflect the trends observed in the soil and air. Figure 7 reflects the validity of this suspicion. Vegetation lead levels also show a distinct relationship with distance from the reference point. Because these are all samples of grasses, washed and unwashed vegetation are grouped in this figure. There is no clear relationship between washed and unwashed grasses except in areas where the airborne particulate matter is heavy with lead.. It is suspected that because of the relatively low exposure of surface area in grasses, the variance among individual grass samples is much greater than the difference observed between washed and unwashed samples.

SUMMARY OF PHASE I

Generally, it is apparent that the center of abnormal lead concentrations in the valley is the Bunker Hill Complex. Other small "hot spots" exist, notably around old mining and milling areas and the flood plains of creeks that service the same. Leaching and wind blown dusts from these areas still constitute an active source. Concentrate is still spilled by trains moving up and down the valley. However, in both magnitude and source strength, the largest and certainly what is expected to be the most significant active source of contamination is air-born emissions from the Bunker Hill Complex. Nevertheless, it must be recognized that the relative public health significance of current emissions from the Bunker Hill Complex and the lead deposited in the environment during past years and via other mechanisms has not yet been established.

SUMMARY OF PHASE III RESULTS

Tables IV and V contain a summary of all samples that have been analyzed to date. As can be seen, the blood lead levels follow the same general pattern as the air, soil and vegetation samples of Phase I (see Figure 8). The lead concentrations found in Areas I and II are significantly high to warrant concern. However, a cursory examination of the data shows no obvious correlation between levels of environmental lead and levels of blood lead in children on an individual basis. This is to be expected, however, since nearly all of the blood lead levels reported for Area I and II are high and the range of values is not great. Therefore, the possibility of correlation of blood lead data in Areas I and II is limited. A more comprehensive examination of Area I and II data may show subtle correlations. Later analysis of all samples from all geographic areas (which include a

greater range of blood lead levels) may show obvious correlations and develop a cause-effect relationship. It is obvious from the data thus far presented that a combination of two or more environmental factors may be influencing the blood lead levels in children.

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POSTSCRIPT

A great deal of information has been collected since May of this year. Many samples have been collected and processed. Many hours of study, investigation, and preparation have been invested. Both the staff and facilities of the Idaho Department of Health and Welfare have been taxed to accomplish the wealth of information acquired. Since the blood lead levels were made public, many agencies have either offered their assistance or initiated studies of their own. Information that has been presented in this paper and information made public thus far represents only limited and incomplete analyses. This has been only a first installment. As more data is processed and analyzed, more information will be released in other forums.

It is only proper that the analyses performed on the data and conclusions drawn from those analyses be accomplished with the same care and the same quality of work as that employed in the collection of that data. Unfortunately, this requires some time. However, for the good of all concerned, it is best to come to the proper conclusions by comprehensive examination than it is to err prematurely.

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REFERENCES

Baltrop, D., "Sources and Significance of Environmental Lead for Children," Paper presented at International Symposium on Environmental Health Aspects of Lead, Amsterdam, October 2-6, 1972.

Carnow, B. and Rosenblune, B., "Unsuspected Community Lead Intoxication and Emissions From a Smelter," Paper presented at 66th Annual Meeting of Air Pollution Control Association, Chicago, Illinois, June 26, 1973.

Environmental Protection Agency, "EPA's Position Paper on the Health Effects of Airborne Lead," Health Effects Branch, Office of Research and Monitoring, Washington, D.C., November 29, 1972.

National Academy of Science, Biological Effects of Atmospheric Pollutants, National Research Council-National Academy of Sciences, Airborne Lead in Perspective, Washington, D.C., 1972.

Needleman, H. and Scanlon, J., "Getting the Lead Out," New England Journal of Medicine 288:466 (1973).

Vostal, J., et.al, "Lead Analysis of House Dust: A Method for the Detection of Another Source of Lead Exposure in Inner City Children," Environmental Health Perspectives, p. 91, May 1974.

TABLE III

SUPPLEMENTARY HEAVY METAL SCREEN - PHASE I (ppm)

Sample #	Type	Cadmium Cd	Arsenic As	Mercury Hg	Zinc Zn	Antimony Sb	
2	Soil	61	72.1	48.1	5953	-	
3	Soil	9	10	<1.5	110.5	-	
5	Soil	8	9	<1.5	1840	-	
10	Soil	-	-	-	-	82	
14	Soil	-	-	-	-	366	
17	Soil	-	-	-	-	66	
19	Soil	-	-	-	-	78	
21	Veg	3.5	<1.2	<1.5	83	-	(washed)
21	Veg	4.0	<1.2	<1.5	89	-	(unwashed)
21	Soil	2.6	10	<1.5	110	-	
22	Soil	-	-	-	-	52	
25	Soil	-	-	-	-	66	
25	Veg	<2	<1.2	<1.5	61	-	(washed)
25	Veg	3	<1.2	<1.5	98	-	(unwashed)
26	Veg	46	<1.2	<1.5	123	-	(unwashed)
26	Veg	<2	<1.2	<1.5	100	-	(washed)
26	Soil	4.5	8.0	<1.5	155	-	
28	Soil	-	-	-	-	52	
29	Soil	2.5	8.6	<1.5	160	-	
33	Soil	3.9	9.0	<1.5	219	-	
37	Soil	10.1	34.6	<1.5	1100		
40	Soil	-	-	-	-	72	
43	Soil	-	-	-	-	60	
44	Soil	4.2	11.8	<1.5	160		
45	Soil	-	-	-	-	64	
47	Soil	-	-	-	-	276	
50	Soil	-	-	-	-	60	

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TABLE IV

AREA I -- Smelterville

	AVERAGE	RANGE
Soil	6,700 ppm	1,089 - 26,700 ppm
Vegetation (grass)	1,200 ppm	109 - 2,200 ppm
House dust	12,000 ppm	900 - 38,900 ppm
Inside paint	-	48 - 179,000 ppm
Outside paint	-	1,047 - 353,000 ppm

AREA II -- Kellogg - Pinehurst

	AVERAGE	RANGE
Soil	3,750 ppm	300 - 18,400 ppm
Vegetation		
House dust	8,700 ppm	2,000 - 49,800 ppm
Inside paint		4 -
Outside paint		

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TABLE V

MICROGRAMS LEAD 100 ml/blood	NUMBER OF BLOOD SAMPLES	PERCENT
Over 80	40	3.8
40-79	388	37.1
0-39	619	59.1
TOTAL TESTED	1,047	
AREA I -- Smelterville		
Over 80	37	21
40-79	136	78
0-39	2	1
Total	175	
AREA II -- Kellogg - Pinehurst		
Over 80	3	1.5
40-79	146	74.5
0-39	47	24.
TOTAL	196	
AREA III		
Over 80	0	
40-79	47	24.7
0-39	143	75.3
TOTAL	190	
AREA IV -- Osburn		
Over 80	0	
40-79	35	21.3
0-39	129	78.7
TOTAL	164	
AREA V -- Mullan		
Over 80	0	
40-79	19	9.7
0-39	175	90.3
TOTAL	194	
AREA VI -- Spirit Lake		
Over 80	0	
40-79	1	1
0-39	88	99
TOTAL	89	
AREA VII -- Pinehurst		
Over 80	0	
40-79	4	10.3
0-39	35	89.7
TOTAL	39	

AVERAGE QUARTERLY LEAD & PARTICULATE DATA

(Kellogg-Smelterville Average)

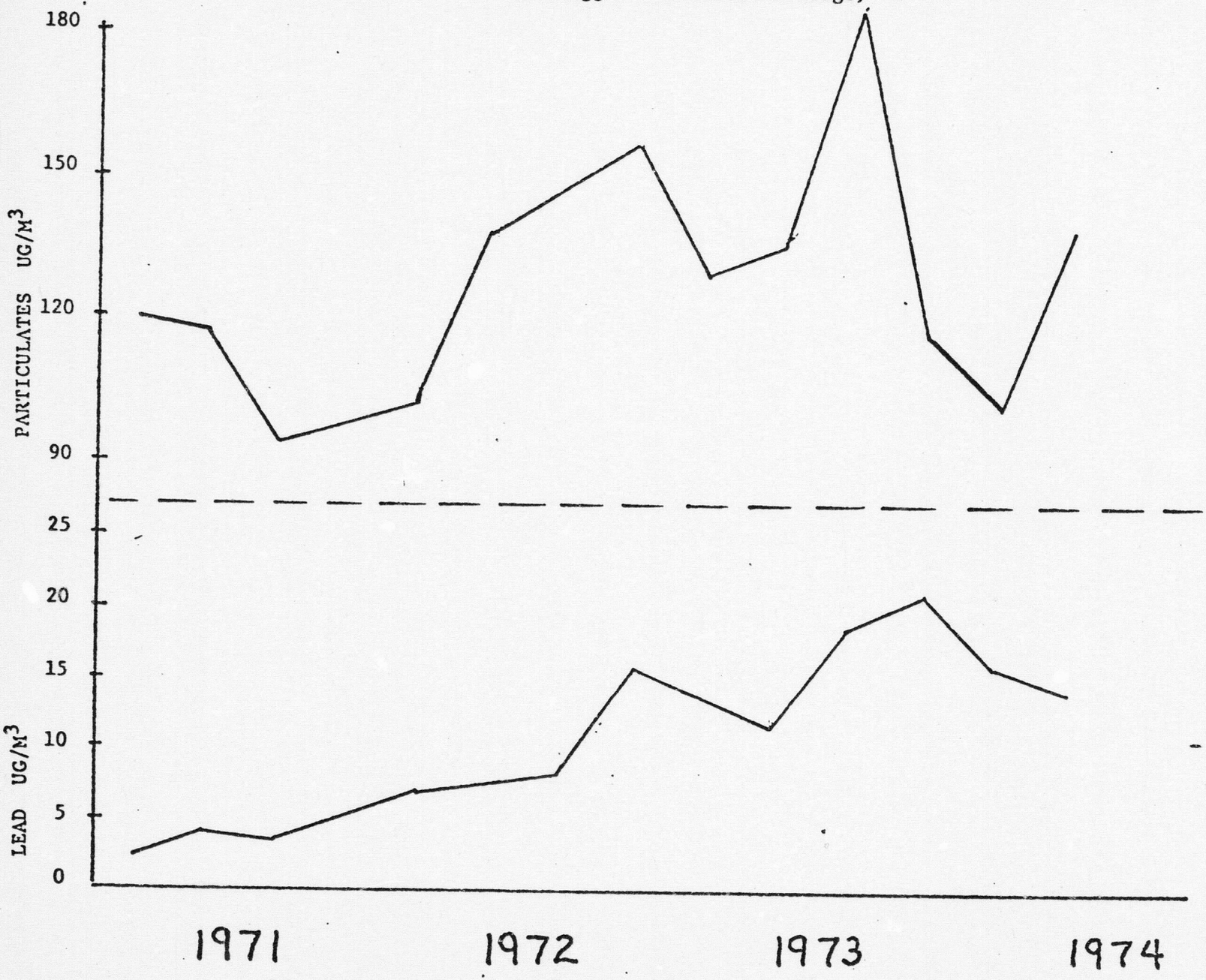


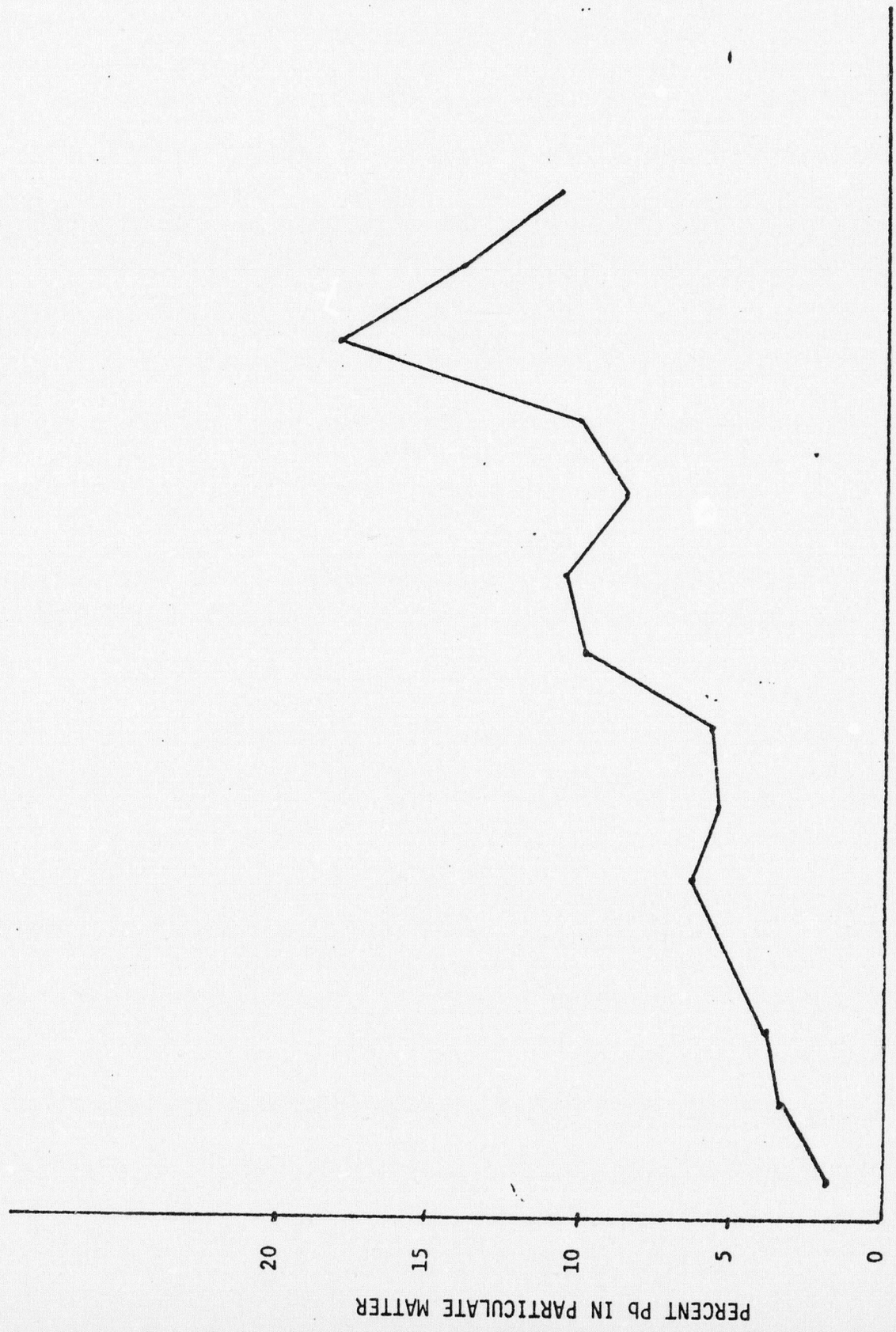
FIGURE 1

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FIGURE 2

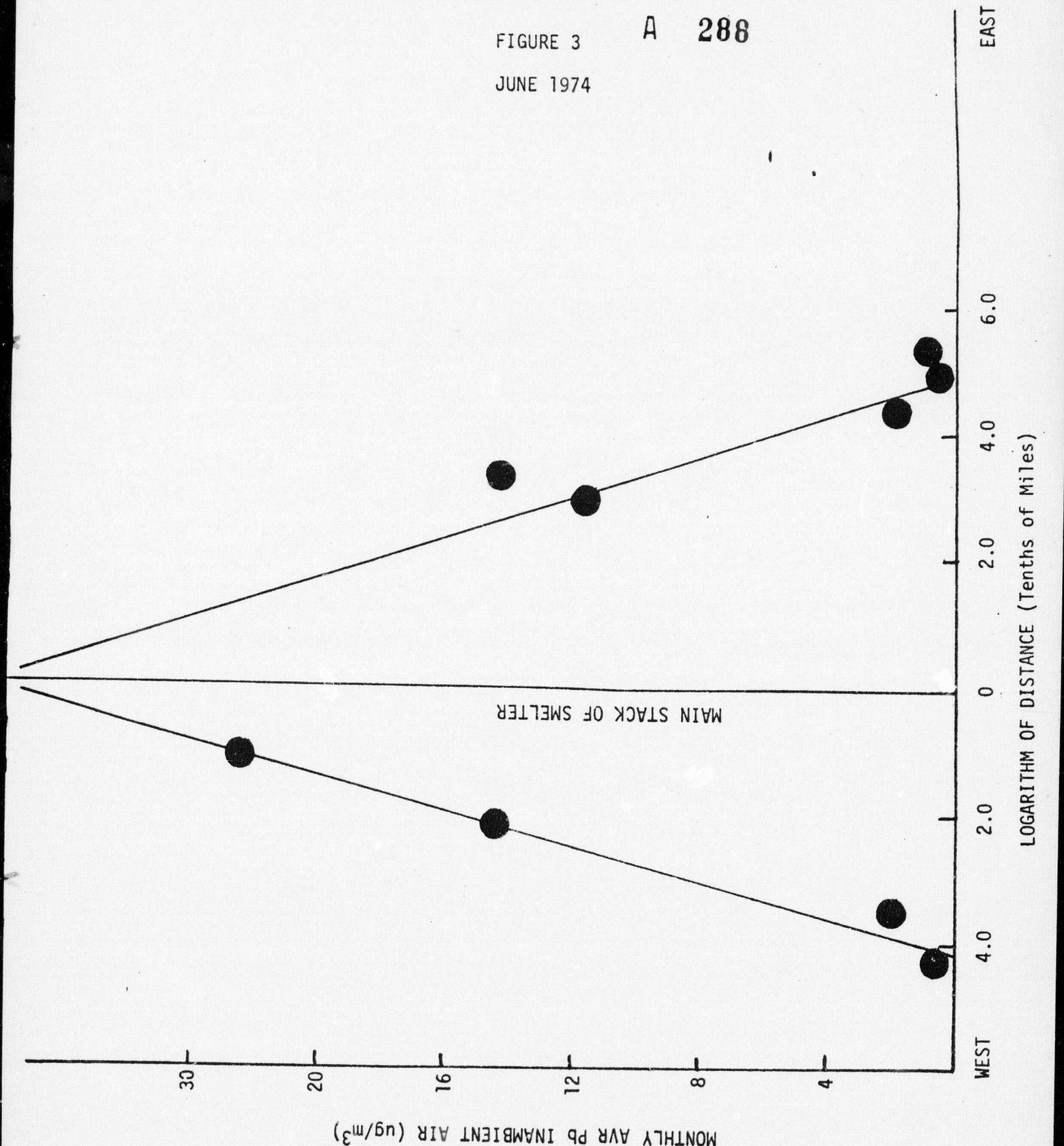
PERCENT Pb IN PARTICULATE (Kellogg-Smelterville Average)
(by Quarter)



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FIGURE 3

JUNE 1974



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FIGURE 4

JULY 1974

FIGURE 5

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AUGUST 1974

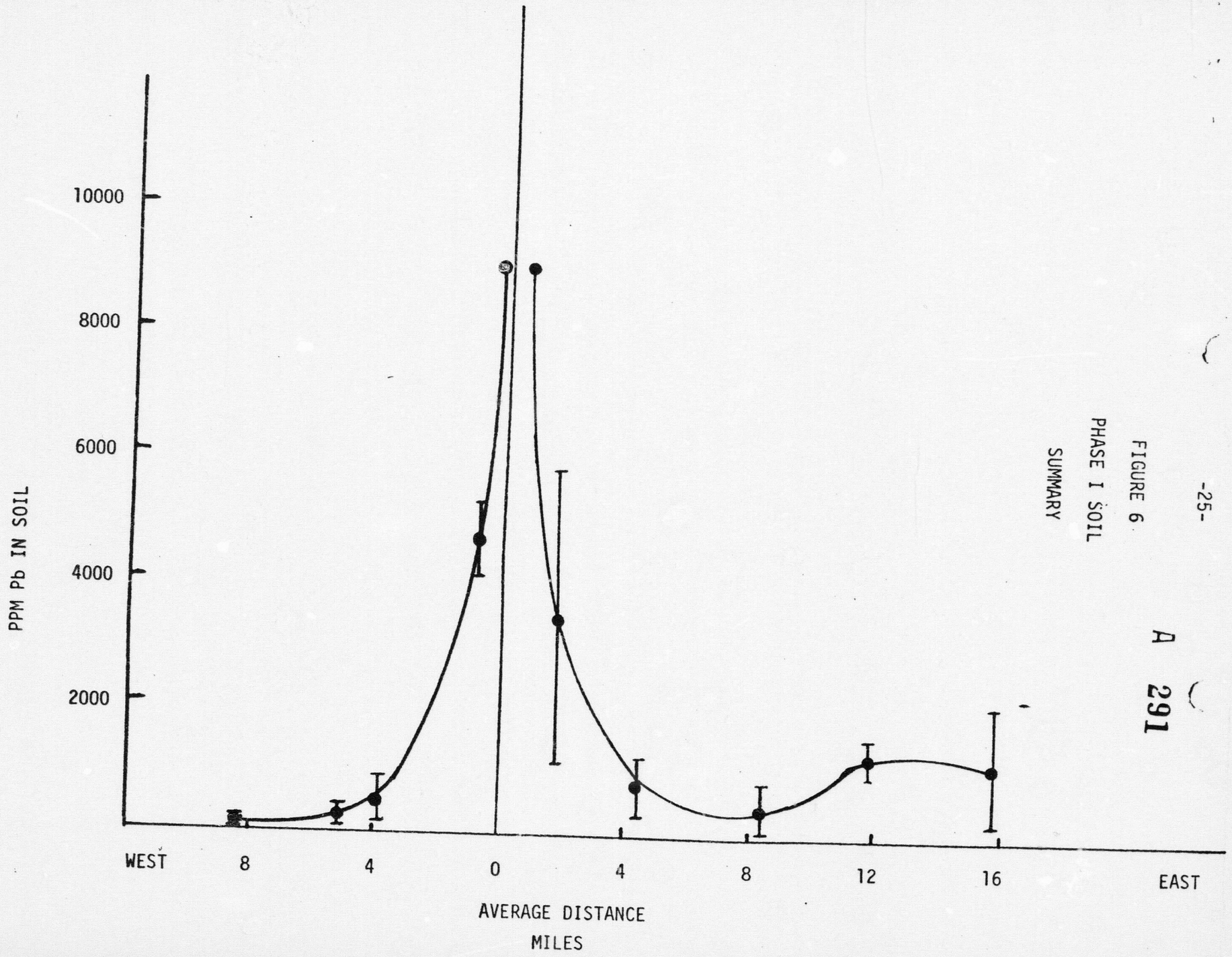
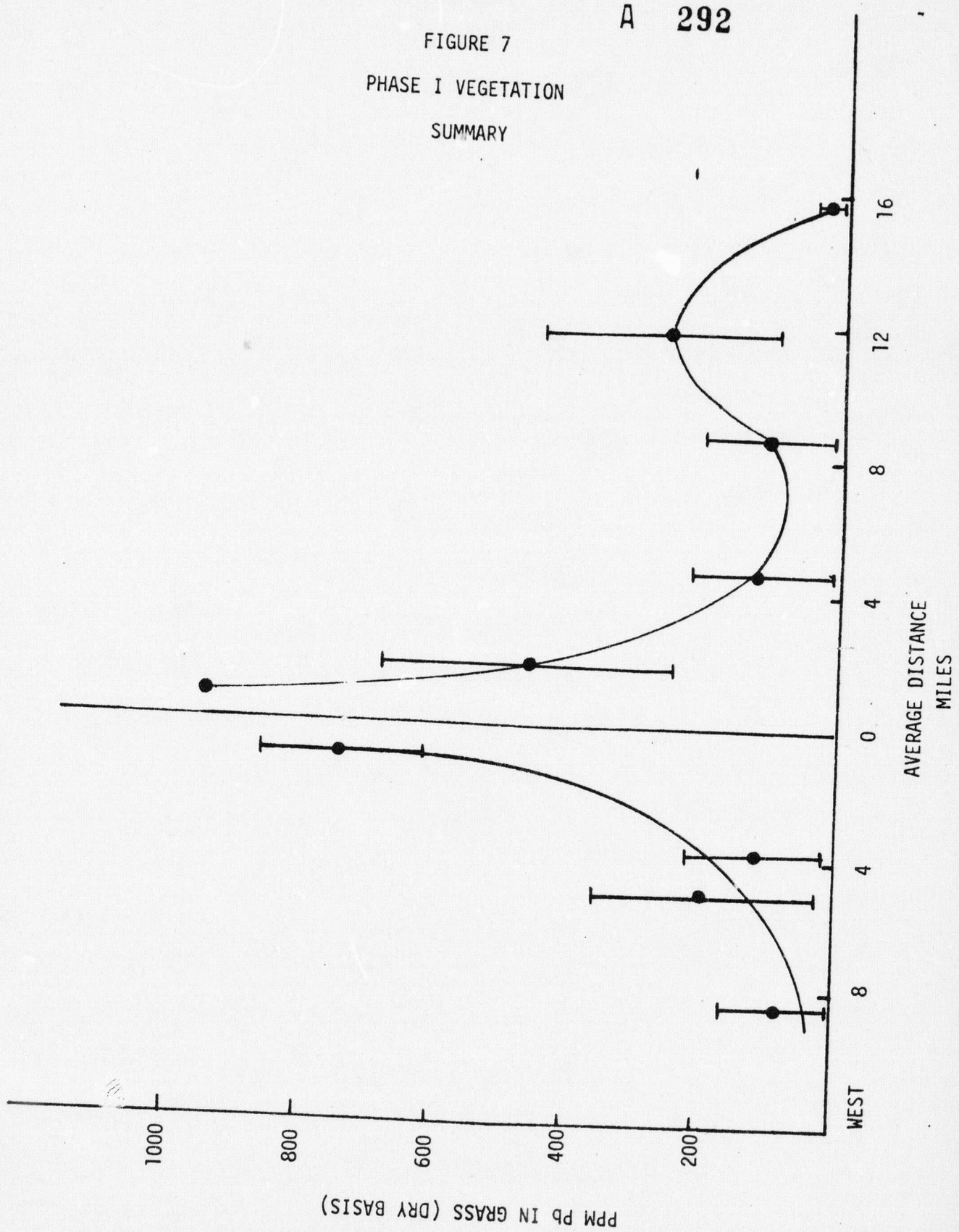


FIGURE 6
PHASE I SOIL
SUMMARY

FIGURE 7

PHASE I VEGETATION

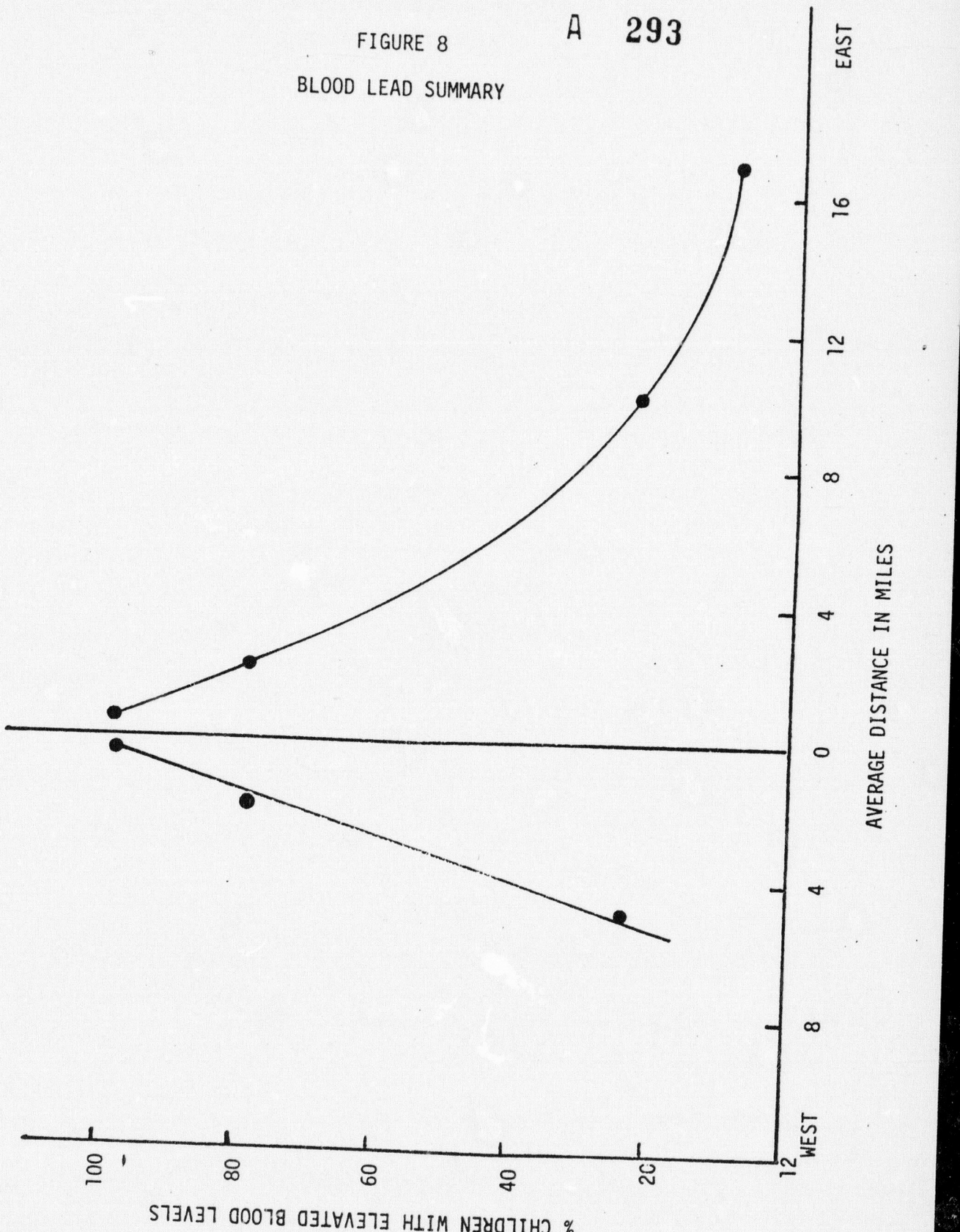
SUMMARY



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FIGURE 8

BLOOD LEAD SUMMARY



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UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
Research Triangle Park, North Carolina 27711
National Air Quality Criteria Advisory Committee
of the Science Advisory Board

November 21, 1974

Mr. Russell E. Train
Administrator
Environmental Protection Agency
Washington, D. C. 20460

Dear Mr. Train:

As Chairman of the National Air Quality Criteria Advisory Committee which is one of the committees constituting the Science Advisory Board and which, under its charter, i.a. provides advice and consultation regarding air quality criteria for specific pollutants, I would like to bring the following to your special attention.

At several recent meetings, the Committee has been concerned with the problem of airborne lead. The Committee has been consulted on the determination and documentation of adverse effects on the public health and welfare of airborne lead from stationary sources and the Committee has been briefed on and has discussed recent research findings on the health implications of airborne lead as well as some of the regulatory aspects of the problem.

The Committee is not satisfied that the public health and welfare can be adequately protected from any known or anticipated adverse effects associated with the presence of lead in the ambient air without the development of ambient air quality criteria and ambient air quality standards. At its most recent meeting on November 14, 1974, the Committee expressed this concern by adopting a motion recommending, for your consideration, that lead be listed as a pollutant, under section 108 of the Clean Air Act, as amended, for which you plan to issue air quality criteria for the purpose of establishing national ambient air quality standards.

I would welcome an early opportunity, for me and some of my colleagues on the Committee, of presenting to you and other appropriate officials the reasons for the Committee's concern.

Sincerely yours,

Arie J. Haagen-Smit
Chairman

Exhibit B

cc: Deputy Administrator
Regional Administrators
Assistant Administrator for Research and Development
Assistant Administrator for Air and Waste Management
Assistant Administrator for Water and Hazardous Materials
Assistant Administrator for Enforcement and General Counsel
Director, Office of Toxic Substances
Dr. Mrak
Dr. Bath
Dr. Wiser
Dr. Buckley
Dr. Steigerwald
Mr. Goodwin
Mr. Padgett
Dr. Finklea
Dr. Hueter
EPA Committee Management Officer
Members, NAQCAC
Executive Secretary, NAQCAC

Natural Resources Defense Council, Inc.

A 296

15 WEST 44TH STREET
NEW YORK, N.Y. 10036
212 869-0150

Washington Office
917 15TH STREET, N.W.
WASHINGTON, D.C. 20005
202 737-5000

Western Office
664 HAMILTON AVENUE
PALO ALTO, CALIF. 94301
415 327-1080

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John H. Adams
Executive Director

December 12, 1974

Mr. Michael T. Scanlon
Administrator's Environmental
Group Liaison
Office of the Administrator
Environmental Protection Agency
Washington, D.C. 20460

Dear Mr. Scanlon:

I am writing to request that you call my enclosed letter to Mr. Train of this date to his personal attention. David Hawkins said that you could do this.

I would like Mr. Train to read this letter because I believe that he has received poor advice on the use of the ambient standard setting process for lead and other pollutants. His response to me of November 23, 1974, does not appear to be based on his own reevaluation of this situation and repeats errors coming from lower levels. The Agency's decision to avoid using the health protection procedures of sections 108-110 of the Clean Air Act (ambient air standards) is a major policy choice that deserves critical reevaluation at the top level, regardless of the outcome in the lead lawsuit now pending. This, in essence, was also the conclusion, without dissent, of the National Air Quality Criteria Advisory Committee as reflected in Dr. Haagen-Smit's letter to Mr. Train of November 21, 1974.

I am enclosing my previous correspondence with Mr. Train and Dr. Haagen-Smit's letter.

Sincerely,

David Schoenbrod

David Schoenbrod

S/t
Enc.

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Exhibit C

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A 297

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Executive Director

December 12, 1974

Russell E. Train, Administrator
U.S. Environmental Protection Agency
Washington, D.C. 20460

Dear Administrator Train:

Thank you for your letter of November 25, 1974, in response to my letter urging a treatment of lead as a criteria pollutant under the health protection procedures of the Clean Air Act (sections 108-110). Quite apart from the legal arguments to be presented in court, I want to reply to you personally to explain why I believe that EPA is making a poor policy choice in disregarding the health protection procedures.

At the outset, it is well to correct EPA's misunderstanding of the effect of using sections 108-110. These sections, read together, provide a mechanism to set mandatory goals, in terms of ambient air concentrations, and strict timetables for achieving these goals. State implementation plans are then, as the Senate Report states (p. 12), only the "principal component" of efforts to meet the goals. As your letter points out, other emission control techniques can be used to achieve these goals. Your letter and other EPA documents falsely assume, however, that setting ambient goals locks the Agency into achieving the goals through state implementation plans, not other federal emission control techniques. Thus, you erroneously see NRDC urging "ambient standards as a control strategy (p. 7)." Rather, NRDC seeks ambient standards for lead as a goal that can be achieved through state implementation plans, section 211, or whatever other emission control technique EPA chooses that will do the job. Specifically, ambient standards could have been set for lead and, as far as mobile sources are concerned, achieved entirely through federal fuel additive regulations. The lack of a goal allowed inadequate control of lead from mobile sources and no control of lead from stationary sources.

Goal-setting should be of considerable importance to the Administrator of an Agency growing beyond its infancy

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since, as numerous studies have concluded, agencies usually seem to go directly from infancy to senility. Agencies without clear legislative goals soon suffer a loss of direction and morale. In this condition, their movement is easily warped and delayed by large economic interests.

Recognizing these problems, agencies without clear legislative mandates have tried to construct goals administratively using various planning tools similar to the Preferred Standards Path Analysis. The Preferred Standards Path Analysis is, however, a poor substitute for the legislative mandate that EPA already has. First, a legislative mandate, in itself, carries far more weight than some charts drawn by internal planners. Second, the Preferred Standards Path Analysis does not subject the Agency to a timetable of action.

In practice, these differences mean a lot, as can be seen in the case of lead. As any involved person in the Agency would admit, EPA's efforts to regulate lead additives under the loose language of section 211 of the Clean Air Act was delayed for years by outside pressure and internal confusion. While there was considerable disagreement between EPA and other groups, the real question inside EPA was what standard of health protection would the Agency be allowed to apply. After many people-years of effort and much rancor, the Agency ended up with almost precisely the same regulation as originally proposed, but with the public health losing through the several years of delay. The health protection procedures would have necessitated a quicker decision under an already established health standard. (Sections 108-110).

Control of lead pollution from stationary sources also suffered from EPA's failure to systematically pursue a clear direction. The newest example is the recently discovered situation at Kellogg, Idaho, where 21% of the nearby children have blood leads above 80 and 99% exceed 40. Once discovered, emissions were cut fourfold in a matter of days. But, the poisoning would never have taken place if EPA had used the planning techniques built into the health protection procedures.

As of now, EPA seems bent upon repeating the same mistakes with many other pollutants other than lead. Since enactment, EPA has applied the procedures to only one new pollutant -- nitrogen oxides.

A word should be added about EPA's argument that it would be difficult, if not impossible, to set an ambient air standard for lead. Similar arguments can be made for any pollutant. Congress recognized that setting an ambient air standard would be difficult,

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told EPA to do so anyway, and specifically stated that it meld in subsequent knowledge by revising the standards later. In any event, you have been misadvised as to the actual facts surrounding the 2 microgram per cubic meter standard for lead. A reading of the health documents circulating at the end of 1972, including EPA's Position on the Health Effects of Airborne Lead and Administrator Ruckelshaus' Memorandum to OMB, explicitly indicate that the 2 microgram standard had been discredited because it was too high to protect health. The problem was not with setting a standard, but with getting measures sufficient to achieve a health-protective standard through OMB review. The health protection procedures sought, however, to set the goal first and then find a way to achieve it.

You suggest that I discuss this situation with Dr. Steigerwald. Because of my view that putting forth the Preferred Standards Path Analysis does both the Agency and public health a disservice, I wonder if such a discussion at this juncture would be useful to Dr. Steigerwald, myself, or the Agency. At the same time, however, I note that Dr. Haagen-Smit, Chairman of the National Air Quality Criteria Advisory Committee wrote you on November 21, 1974, advising you of the Committee's resolution asking you to list lead and asking you for a meeting to discuss the matter with you. I would very much appreciate being invited to such a meeting, if Dr. Haagen-Smit would not object. The latter meeting, in my view, would be more clearly aimed at reaching a lead control strategy which, as your letter notes, "affords the most effective means of achieving the reduction in lead emissions we both desire."

Sincerely,

David Schoenbrod

David Schoenbrod

S/t
cc: Dr. Haagen-Smit